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LEPTOTRICHOSIS CONJUNCTIVAE (PARINAUD'S CONJUNCTIVITIS)

ARTIFICIAL CULTIVATION OF THE LEPTOTRICHES IN THREE OF FOUR CASES

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In previous communications [1] the senior author has shown that there is a disease of the conjunctiva accompanied by massive inflammatory enlargement of the preauricular and sometimes of other regional lymph glands which has certain clinical and histologic features so characteristic that either suffices to establish the disease as an entity. The essential conjunctival lesions appear clinically as gray or yellowish areas just beneath the epithelium, and histologically as localized infiltrations of the tissues with macrophages loaded with broken-down chromatin. The inflammatory reaction causes the conjunctiva to become elevated into nodules or ridges. The latter have often inaccurately been described in the literature as granulations.

In 1913, the senior author, by a special method of staining, demonstrated a minute leptothrix within the conjunctival lesions. This was present only within the essential lesions; it occurred in great numbers, was accompanied by no other demonstrable organisms and was present in every case studied. For these reasons it was regarded as the cause of the disease, and the term leptotrichosis conjunctivae was suggested as the most suitable designation for the latter. The organism has now been found in forty-five instances, comprising, with one exception, all cases of the disease encountered at the Massachusetts Eye and Ear Infirmary within the past twenty-eight years. Elsewhere, so far as we know, the only investigator who has been able to find the organism is Sanford R. Gifford.[2] The staining method is extremely delicate—if the differentia-

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1. Verhoeff, F. H., and Derby, G. S.: Arch. Ophth. **33**:389, 1904; Klin. Monatsbl. f. Augenh. **43**:18, 1905. Verhoeff, F. H.: Arch. Ophth. **42**:345, 1913; Am. J. Ophth. **1**:705, 1918.

2. Gifford, S. R.: Am. J. Ophth. **10**:484, 1927.

tion is continued for even a few seconds too long, the organisms may be completely decolorized.

In 1889, Parinaud described briefly three cases of an unusual disease characterized by the formation, in the conjunctiva, of granulations, as he called them, and by inflammatory enlargement of the preauricular gland. He did not set forth the clinical features in sufficient detail to establish a clinical entity, and he did not investigate the cases histologically. He also failed to mention the gray areas in the conjunctiva which are the essential lesions of leptotrichosis of the conjunctiva. Nevertheless, his cases were evidently so similar to those investigated by the senior author that there is no reasonable doubt but that they were of the same nature. Since Parinaud's communication many cases have been reported in the literature as Parinaud's conjunctivitis. Exclusive of those definitely proved to be tuberculous, most of the cases were probably true instances of the disease, although the majority were so inadequately described or investigated as to be doubtful when considered individually.

Tularemia of the conjunctiva can easily be distinguished from Parinaud's conjunctivitis, and in any case is too rare to have caused much confusion. Pascheff's disease is probably identical with it, as was pointed out by Vail.[3] In this vicinity no case of tularemia of the conjunctiva has been recognized, and Parinaud's conjunctivitis has been much more frequent than conjunctival tuberculosis. During the period when forty-five cases of Parinaud's conjunctivitis were encountered there were only thirteen cases of conjunctival tuberculosis, including three cases of lupus. In none of the cases of Parinaud's conjunctivitis studied by the senior author did ulceration of the conjunctiva occur. While it is, of course, possible that ulceration might occur, it is probable that in most of the cases in the literature in which this was described areas of infiltration were mistaken for actual ulcers.

Hitherto no one has succeeded in securing the organisms in culture from the conjunctival lesions of Parinaud's conjunctivitis. In 1918, Wherry and Ray,[4] in a supposed case of this disease, cultivated a leptothrix from the preauricular gland. After two weeks, under conditions of partial tension, they obtained a feeble growth on Dorset's egg medium. No microscopic examination of the conjunctival lesion was made, and hence no attempt was made to demonstrate the organisms in these lesions. Judging from their observations alone, it would seem, therefore, to be extremely doubtful that the organisms grown by them were the causative factors in Parinaud's disease. Since, however, leptotriches have been so rarely cultivated from any source; since we know that such organisms are the cause of Parinaud's conjunctivitis, and since,

3. Pascheff, C.: Klin. Monatsbl. f. Augenh. **57**:517, 1919. Vail, D. T., Jr.: Oculoglandular Form of Tularemia, Arch. Ophth. **2**:416 (Oct.) 1929.

4. Wherry, W. B., and Ray, V.: J. Infect. Dis. **22**:554, 1918.

as will be stated, we have been successful in cultivating leptotriches from the conjunctival lesions, we believe it is probable that the organisms recovered in culture by Wherry and Ray from the preauricular gland were actually the causative agents in the disease in question. Wherry and Ray gave an extremely meager description of the organism, and apparently allowed the strain to die out without investigating its cultural characteristics further. They inoculated two white mice with the organisms without securing significant results.

Recently we have succeeded in obtaining leptotriches in pure culture from the conjunctival lesions in three of four cases of Parinaud's conjunctivitis. The four cases showed the typical clinical and histologic pictures of the disease, and leptotriches were found in the lesions in sections. In the unsuccessful case the lesions were small; in the others they were comparatively large.

In each case the correct diagnosis was made clinically by the senior author when the patient was first seen. The three cases in which the leptotriches were cultivated are described briefly.

REPORT OF CASES

CASE 1.—A girl, aged 13, was first seen in the clinic on Oct. 18, 1931. Two weeks previously she had noticed that her left eye was slightly reddened. This redness gradually increased, the eyelids became slightly swollen, and some "tearing" developed. There was no pain and but little discomfort. Attendance at school had been regular, and there was no history of exposure to persons with "sore eyes." She had, however, played with a cat at home.

Examination showed the upper bulbar conjunctiva of the left eye to be congested and elevated over an area of considerable size, which extended to the fornix. Within this area, just beneath the surface, were three small, yellowish-white, nonulcerated spots about the size of a pinhead. There was enlargement of the lymph follicles of the palpebral conjunctiva. The cornea was clear. Vision in the right eye was 6/6; that in the left eye, 6/9. The left preauricular node was the size of a hickory nut, and the anterior cervical nodes were slightly enlarged. Constitutional symptoms were entirely lacking. Smears from the conjunctival secretion showed no micro-organisms. The conjunctival lesion was excised for laboratory studies. The conjunctiva healed promptly, and the ocular congestion decreased, but for two weeks the preauricular node enlarged slowly to diameters of about 2 by 2 by 1.5 cm., and the cervical nodes became pea-sized. The temperature rose to 99.4 F. for several days. The glands never "broke down" and slowly decreased in size, and about one month after the patient's first visit to the clinic her preauricular node was only about the size of a marble. She made an uneventful recovery.

CASE 2.—A boy, aged 5½, was brought to the clinic because the right eye was red and congested; the condition had existed for one week. He had never been to school. He had played with two kittens at home.

On eversion of the edematous upper lid, the upper bulbar conjunctiva was seen to be congested and elevated over an area about 8 by 4 mm. in size, with a grayish-white focus in the center. There was marked enlargement of the ordinary lymph follicles of the palpebral conjunctiva. The cornea was unaffected. There was a

slight amount of secretion, but smears showed no micro-organisms. The right preauricular lymph node was the size of a marble and tender to pressure, but there was no palpable enlargement of the cervical or maxillary nodes. The conjunctival lesion was excised for laboratory studies. The conjunctiva cleared promptly, the preauricular node slowly decreased in size, and six weeks after the first visit to the clinic all signs of the infection had disappeared.

CASE 3.—A boy, aged 3½, was first seen on Jan. 15, 1932, at the clinic. For the past three weeks the right eye had been "red and swollen," and the day before admission the parents noticed that the "gland in front of the right ear was enlarged and tender." Until one month before admission the child had played with two kittens and a mother cat which were kept in the home.

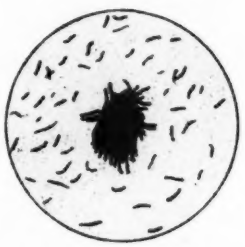

Fig. 1 (case 1).—Initial growth. Verhoeff's modified Gram stain; serum-agar one week at 37 C. × about 1,200.

Fig. 2 (case 2).—Clubbing, long and short filaments. Methylene blue (methylthionine chloride, U. S. P.), serum-agar three weeks at 37 C. × about 1,200.

Examination showed five grayish-white areas in a congested and elevated portion of the upper bulbar conjunctiva just below the fornix on the temporal side. The largest of these grayish areas was about 2 mm. in diameter. There was enlargement of the lymph follicles of the palpebral conjunctiva. The right preauricular node was as large as a horse-chestnut and was tender to pressure. Smears of the mucoid conjunctival secretion showed no micro-organisms. The main conjunctival lesion was excised for laboratory studies. The parents did not bring the boy back to the clinic until eight weeks later, when they stated that after excision of the lesion the eye had gradually whitened, and that the preauricular node had first enlarged slightly but soon gradually decreased in size. At the end of six weeks, they stated, they could not feel the node.

In each of the cases half of the excised tissue was fixed in Zenker's fluid, and sections were later stained in hematoxylin and eosin and by

Verhoeff's modified Gram stain. Histologically, the lesions were identical in appearance with those previously described by Verhoeff, and in each case leptotriches were revealed by the special stain.

BACTERIOLOGY

In each case the conjunctival lesion was excised under aseptic precautions. A portion of each specimen was macerated in a sterile mortar and emulsified with sterile physiologic solution of sodium of chloride. Direct smears were made of the emulsions, as well as cultures and ani-

Fig. 3 (case 3).—Interlacing filaments. Carbolfuchsin; serum-agar two weeks at 37 C. × about 1,200.

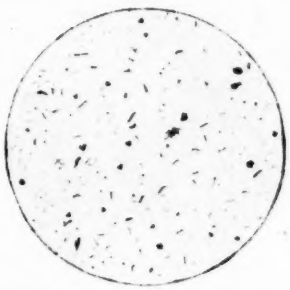

Fig. 4 (case 3).—Extruded coccoid bodies. Carbolfuchsin; serum-agar two months at 22 C. × about 1,200.

mal inoculations. The direct smears in cases 1 and 3 showed a few leptotriches. Most of these were entirely gram-negative, but a few partially retained the gentian-violet. The emulsions were planted on a variety of mediums and incubated under aerobic and anaroebic conditions and in an atmosphere containing 10 per cent carbon dioxide. Leptotriches were cultivated in pure form.

Initial growths of the organism occurred only on dextrose-serum-agar and serum-agar, and only at 37 C. in jars in which 10 per cent of the air was replaced by carbon dioxide. In cases 1 and 2 the serum was obtained from guinea-pigs and in case 3 from a human being. The growth was slow, appearing in about six days as minute, discrete, gray-

ish-white, elevated, adherent colonies. After from three to six subcultures the organisms grew faster, became less fastidious and could be cultivated on most of the usual laboratory mediums without the presence

Fig. 5 (case 2).—Long filaments. Carbolfuchsin-serum-broth two days at 37 C. × 900.

Fig. 6 (case 2).—Early spore formation and short filaments with granules. Moeller's stain; serum-agar two weeks at 22 C. × 700.

Fig. 7 (case 2).—Abundant spore formation. Methylene blue; serum-agar three weeks at 22 C. × 1,000.

of carbon dioxide. The adherent characteristic was retained after months of subculturing.

The organisms were gram-negative and nonacid-fast and stained faintly in the usual aniline dyes. They were best seen when stained quickly in carbolfuchsin. The strains from all three cases were essen-

tially alike, but in case 1, in the early cultures, there was a larger proportion of the smallest forms than in the other two cases. The organisms in general were larger than those seen in the sections, and they failed to stain by Verhoeff's modified Gram method. In the initial culture in case 1, however, they could be stained by the modified Gram method so as to give the exact appearance of those in the sections.

The organisms were pleomorphic, varied greatly in size and in the young cultures consisted of large and fine filaments, as well as bacillary forms, and, as a rule, were more or less curved. The older cultures were represented mainly by faintly staining sheaths containing more deeply staining granules. Still older cultures showed what appeared to be ghost forms in which the sheaths stained scarcely at all; the filaments appeared to have disintegrated and liberated fine gram-negative granules —the so-called coccoid bodies. In case 3 spore forms were demonstrated in the original cultures. In the other two cases the spores did not form until after several transplants had been made at a temperature of from 22 to 24 C. Cultures grown at room temperature developed mainly as coccoid and spore forms, with infrequent bacillary types. Subcultures at this stage, left at 37 C., slowly reproduced the original forms, consisting of gram-negative bacilli and longer filaments. In from three to four days the filaments tended to break down by septal division into shorter segments and bacilli. In the smallest forms, septal divisions were not seen. In the initial cultures, in case 1 all the organisms were of about the same width, 0.3μ. The shortest was 2μ, and the longest, 20μ. In case 2 they were about 0.5μ in width; the shortest was 4μ, and the longest, 37μ. In case 3 the width was 0.6μ; the shortest length was 6μ, and the longest, 101μ.

In general, few spores were produced at 37 C., but in fourteen days, at room temperature under aerobic conditions, numerous spores were formed in serum mediums, and in twenty-eight days spores and coccoid bodies greatly predominated. They developed chiefly in the smaller filaments and always singly at one end. For a time a thin remnant of the filament remained connected with the spore, presenting the appearance of a spermatozoon. They were ovoid and varied little in size, about 2 by 1μ. In the earliest stage it was difficult to distinguish them from coccoid bodies. Newly formed spores stained by Moeller's method showed a typical morphology. Transplants of the spores required incubation for nine days at 37 C. before germination could be observed; then minute filaments could be seen extending at one end and occasionally at both ends of the spores. Growth was obtained from the spore at the stage of 3 months of age. Young filamentous and bacillary cultures were killed at a temperature of 60 C. in half an hour, whereas cultures grown for from twenty to thirty days at 22 C., containing many spores, resisted 80 C.

In a forty-eight hour serum-agar culture at room temperature the organisms were actively motile. After four days the motility was much reduced. The filaments did not undulate, suggesting the presence of flagella. When a spore was present, the organism moved forward at the spore end. Loeffler's stain for flagella made the organisms appear to be much wider, but revealed no definite flagella.

CULTURAL CHARACTERISTICS

These were as follows:

(*a*) *Solid Mediums.*—After organisms had been subcultured:

1. Dextrose-Serum-Agar: appearing in two days as minute, grayish-white, pinpoint colonies; in from four to six days the colonies were about 2 mm. in diameter; elevated, circular in outline and adherent. This property of adherence persisted throughout a long series of subcultures.
2. Dextrose-Agar: similar to 1.
3. Serum-Agar: similar to 1.
4. Blood Agar: similar to 1, but less profuse and less rapid.
5. Coagulated Egg Yolk: similar to 1, but less profuse and less rapid.
6. Blood Serum: no digestion, no discoloration.
7. Plain Agar and Glycerin Agar: growth only after repeated subcultures when the organism had become "medium-wise."
8. Gelatin Stab: slow growth along the stab, with some liquefaction after seven days. Culture from case 3 gave lateral treelike growth.

(*b*) *Liquid Mediums.*—These gave the following results:

1. Hormone broth
2. Vitamin liver broth
3. Chopped kidney

slow, poor growth, forming slight turbidity which fell to bottom of tube as yellowish-white deposit.

4. Dextrose broth: rate more rapid and profuse.
5. Sugar mediums—see table.

Results on Sugar Mediums *

	Case 1	Case 2	Case 3
Dextrose	±	±	+
Lactose	0	0	±
Maltose	±	+	+
Mannite	±	0	0 ±
Rhamnose	0	0	0
Saccharose	0	0	±
Xylose	0	0	0
Litmus milk	0	0	0
Inulin	0	0	0

* 0 indicates no change; ±, very slight acid; ±, slight acid; +, definite acid. Incubated one week aerobically.

EXPERIMENTS ON ANIMALS

We were unable to demonstrate lesions in white mice into which the fresh tissue emulsions and saline suspensions of the organisms cultivated from each case had been injected subcutaneously. Subconjunctival injec-

tions of the organisms into guinea-pigs, kittens and Macacus monkeys consistently produced congestion and swelling of the conjunctiva at the site of the injection, but, with one exception, the swelling and redness began to subside after from twenty-four to forty-eight hours, and had disappeared by the sixth to the ninth day after the injection. In one guinea-pig considerable conjunctival congestion and swelling developed, with a definite yellowish-gray central area, 2 by 1.5 mm., which resembled the lesions in Parinaud's conjunctivitis. This lesion was excised on the ninth day after injection and sectioned.

A somewhat different reaction followed subconjunctival injections into rabbits. Conjunctival congestion and swelling developed in the thirteen animals that were used, but in seven the reaction had subsided in from six to nine days without definite development. In rabbit 1 there developed a swollen area, 5 by 8 mm., in twenty-four hours; the reaction subsided slowly until the seventh day, when a nodule, 2 by 3 mm., was excised. Rabbit 2 showed an indurated nodule, about 2 by 3 mm., within the congested conjunctiva on the ninth day, when the nodule was excised. In rabbit 4 an ovoid nodule, 1 cm. in length and 5 mm. broad, developed within four days. On the fourth day, at the time of the excision of the nodule, there was a small yellowish spot the size of a pinhead at one end of the nodule. In rabbit 3 the usual injection of the organisms under the tarsal conjunctiva was made. Two days later there were marked congestion and swelling of the entire conjunctiva, with a slight whitish secretion. Four days after the injection the swelling and infiltration were subsiding, but a nodule, about 2 by 2 mm. in diameter, had developed. On the sixth day there was less reaction, but the nodule was circumscribed, about 5 by 10 mm., of a grayish-yellow color and considerably indurated. There was no enlargement of the preauricular lymph node. The lesion was excised, and leptotriches were found in direct smears and cultivated from the nodule by the method used for obtaining the initial cultures of the organisms. In rabbit 10 an elevated nodule, 5 by 6 mm., developed within two days. The surrounding conjunctiva was infiltrated and swollen. Three days later the conjunctival swelling had almost subsided, and the nodule was about 1 by 1 mm. in size. After eight days this nodule had disappeared. In rabbit 12 the usual conjunctival congestion, as well as a nodule 5 by 5 mm., had developed in two days. On the sixth day after injection the discrete, yellowish nodule measured 12 by 3 mm., and was excised. In addition to the foregoing experiments, the emulsion of the original material from each patient was injected into the groin of a guinea-pig, and the animals were killed at the end of two months. None of these animals showed any evidences of tuberculosis at autopsy.

Microscopic examinations of the excised nodules from these animals showed the presence of the same lesion in each. This consisted essen-

tially of massive infiltration of the subepithelial tissue with macrophages. Few cells of any other kind were present in this infiltrate. Many of the macrophages were undergoing necrosis, but somewhat fewer cells packed with chromatin fragments were seen than occur in lesions in human beings, due, no doubt, to the much shorter duration of the process in the animals. Around this central infiltration there was a beginning formation of granulation tissue, and a few pus cells were present. In rabbit 4 the surrounding lymph spaces were packed with lymphocytes, among which a number of macrophages could be seen. In rabbit 3, in which the lesion was most massive at the time of excision, the surrounding tissue contained many eosinophils. In the guinea-pig the lesion was extremely small, but consisted chiefly of macrophages, as in the rabbits, and there was little surrounding infiltration. No organisms could be demonstrated in the lesions by special staining, although, as previously stated, leptotriches were recovered in cultures from one of the nodules. Evidently the infection was being overcome before the organisms had an opportunity to resume their original character.

COMMENT

Although no evidence additional to that previously brought forward by one of us (Dr. Verhoeff) is needed to establish the fact that Parinaud's conjunctivitis is due to infection with a leptothrix, our success in cultivating the organisms has revealed certain of their characteristics that could not otherwise have been ascertained. On artificial mediums the organisms become larger and longer, exhibit motility in fresh cultures and finally develop spores. They also become more strongly gram-negative, so that soon they cannot be stained by the modified Gram method, which stains them intensely in tissues. Since in two of the three cases a few of the larger forms were found in direct smears of the emulsified tissues, it is evident that even in the tissues such forms must exist, although, owing to their failure to stain by the methods employed, they cannot be demonstrated in the sections.

By injecting the organisms beneath the conjunctivae of rabbits, we were able to produce ocular lesions closely resembling, clinically and histologically, the lesions of Parinaud's conjunctivitis, but the whole picture of the disease was not reproduced in that glandular involvement was entirely absent and the lesions were of short duration. Failure to produce such experimental lesions by inoculation with excised tissue is explained by the fact that in this way only insufficient doses can be given. The organisms are only slightly pathogenic even to human beings—the disease is never fatal—so that it is not surprising that they are even less pathogenic for animals. It is perhaps noteworthy that the incubation period of the experimental disease was the same—about four days—as that determined by one of us (Dr. Verhoeff) for the disease in human beings.

The term "Leptothriceae" was first used by Kuetzing [5] in 1843. This investigator found the family group of slender filamentous organisms in iron springs and marshy ground. In 1847, Robin [6] first described Leptothriceae buccalis, which he found on the teeth of a horse. Kligler,[7] in 1915, succeeded in cultivating fifteen strains of Leptothriceae buccalis, which he described as thick, long, straight or curved threads, with a clubbed end at one extremity and a tapering end at the other.

According to Lehmann and Neumann [8] (1927), the leptothrix group and the family to which it belongs, Desmobacteriaceae, are more closely allied to the lower bacteria, but they suggest that the species belonging to this family show a closer relationship with the algae than with any of the other lower bacteria. These observers point out that there is no branching. The Committee of American Bacteriologists place Leptothriceae in close relationship to the higher bacteria.

Attempts to define the morphologic and biologic characters precisely have not, as yet, been successful. The difficulty in determining the position and assessing the pathologic importance of Leptothriceae arises largely from the fact that attempts to cultivate these organisms have seldom been successful. Although Leptothriceae are widely distributed in nature, a review of the literature dealing with the organisms discloses so great a discrepancy in respect to staining reactions, biologic characters, motility and spore formation that one is led to regard the subject as in a state of chaos.

As regards the pathogenicity of the organisms, the medical literature contains numerous records of cases in which filamentous organisms have been considered the causal agents of disease. The majority of these comprise a disease of the mouth known as keratosis pharyngis leptothrica, pharyngomycosis, mycosis tonsillaris benigna, etc. Huntmüller [9] (1927) attributed the infrequency of infection to the absence of favorable conditions of growth.

Among the earlier records are those describing inflammation of the lacrimal ducts. In 1869, A. von Graefe [10] published a case in which massed filaments were observed in the inflamed eye. Other accounts of

5. Kuetzing: Phycologia generalis, 1843.

6. Robin: Des végétaux qui croissent sur les animaux vivants, Paris, 1847; cited from Mackenzie.[15]

7. Kligler: A Biochemical Study and Differentiation of Oral Bacteria, with Special Reference to Dental Caries, Dissert., Columbia University, 1915.

8. Lehmann, K. B., and Neumann, R. O.: Atlas und Grundriss der Bakteriologie und Lehrbuch der speziellen bakteriologischen Diagnostik, ed. 7, Munich, J. F. Lehmann, 1927.

9. Huntmüller, in Kolle, Krause and Uhlenhuth: Handbuch der pathologischen Mikroorganismen, Jena, Gustav Fischer, 1927, vol. 5, p. 2.

10. von Graefe, A.: Arch. f. Ophth. **15**:318, 1869.

similar cases have been reported, and it is suggested that the infection of the tear duct arises from organisms originally present in the mouth. We have, however, had the opportunity to examine the concretions in infections of the canaliculi, and have always found the filamentous organism to be a branching form of streptothrix, not the nonbranching leptothrix.

In 1905, Matthews [11] described a case of localized empyema in which the only organism found was a gram-negative leptothrix. He was unsuccessful in his attempts at artificial cultivation. In 1920, Löhlein [12] described the presence of filamentous organisms in ulcers, and in the same year F. J. Lang [13] reported a case of duodenal ulcer, associated with infarcts of the liver, in which the lesions contained abundant gram-negative, nonbranching filaments and cocci. In 1901, Blumer and McFarlane [14] recorded an epidemic of sixteen cases of noma which occurred in the convalescent stage of measles; the condition was attributed to leptothrix infection. Cultural experiments were not made. There are also accounts of meningitis due to filamentous organisms resembling Leptothriceae. Mackenzie [15] cultivated gram-negative, spore-bearing, motile leptotriches—aerobically on blood-agar at 37 C.—from the spinal fluid in six cases of meningitis. In two of the cases the organism was recovered also from the blood in dextrose broth cultures. Of his six patients, two died and four made a perfect recovery; in one of the four a recurrence of the infection took place after an interval of six months, the patient again recovering. It is commonly known that whenever tartar is present around the teeth, filaments of Leptothriceae buccalis are present. In 1924, Bulleid [16] cultivated nonmotile, gram-negative and gram-positive, nonspore-forming leptotriches on saliva blood-agar from tartar around teeth. He found his strains to be facultative anaerobic, and in primary culture development took place more rapidly under anaerobic conditions at an optimum temperature of 37 C. No smear taken from any situation in which tartar was present failed to show the presence of the leptothrix threads, and the organisms were found in the tartar of teeth from rabbits and cats as well as in that from human beings.

We are still in the dark as regards the source of the infection in Parinaud's conjunctivitis. Parinaud's belief that the disease was of animal origin was based on evidence that we now know to be unreliable. It can now be regarded as certain that the infection is not derived from

11. Matthews: Practitioner **74**:197, 1905.
12. Löhlein, W.: Virchows Arch. f. path. Anat. **227**:86, 1920.
13. Lang, F. J.: Virchows Arch. f. path. Anat. **234**:367, 1920.
14. Blumer and McFarlane: Am. J. M. Sc. **122**:527, 1901.
15. Mackenzie: Medical Research Council, London, 1931, vol. 8, p. 90.
16. Bulleid, A.: Guy's Hosp. Rep. **74**:444, 1924.

diseased animals, as he assumed. Whether the leptotriches are of the same kind as those commonly found around the teeth of human beings and of lower animals remains to be determined. In their morphology and cultural characteristics they seem to resemble closely the organisms obtained by Mackenzie in culture from the spinal fluid in his cases of meningitis. The only possible theory as to the source of the infection that has the slightest degree of plausibility is that the infection comes from the mouths of cats. A history of close association with cats has been frequent in the cases from Boston. Although the disease may occur at any season, it has been most commonly seen in the early winter months. Possibly this is due to the fact that cats spend more time indoors during the winter. The great infrequency of the disease may be due to one or more of three facts: first, to the low virulence of the organisms, which would require unusual conditions for infection to take place; second, to a lack of susceptibility of most persons, and third, to the infrequency with which this variety of organism is encountered.

SUMMARY

As previously demonstrated by one of us (Dr. Verhoeff), there is a disease of the conjunctiva associated with inflammatory enlargement of the preauricular gland which is both a clinical and a histologic entity and which is due to infection with a leptothrix. This disease is commonly known as Parinaud's conjunctivitis, although Parinaud did not describe his three cases in sufficient detail to establish them as a complete entity.

In three of four recent cases of the disease we have succeeded in obtaining the organisms in pure cultures on artificial mediums. In each of these cases the clinical and the histologic features were typical, and by special staining leptotriches were demonstrated in the tissues. Special mediums and conditions of partial oxygen tension were required for the first growths, but when once obtained the organisms grew fairly well on ordinary mediums.

Inoculations of the organisms into the conjunctivae of rabbits and of guinea-pigs produced lesions clinically and histologically similar to those of the disease in human beings. The lesions, however, quickly disappeared, and were not associated with enlargement of the regional lymph glands. The one attempt made to recover the organisms from an experimental lesion was successful.

DISCUSSION

DR. MARK J. SCHOENBERG, New York: A few months ago Dr. Dwyer of New York reported two cases of tularemia, and showed pictures of these cases. He made the statement that he believed that Parinaud's conjunctivitis was nothing but tularemia. Dr. Dwyer is a bacteriologist, and I have great respect for his

views regarding bacteriologic methods. Recently, I saw a case of swelling of the lids with changes in the conjunctiva, which made me think that it was somewhat similar to the cases Dr. Dwyer described. I sent the patient to Dr. Dwyer, who made smears and found what he thought was a micro-organism similar to that in the cases he described. A culture could not be obtained. Blood was sent to Washington, but the test was negative. Since clinically the case was similar to those seen before, it seemed that the condition was tularemia

Dr. F. H. Verhoeff, Boston: In all of the cases I have seen of what we call Parinaud's conjunctivitis, I have never seen any involvement of the cornea. I have not examined any of the patients with the slit-lamp, but the corneas seemed perfectly normal, and never at any stage showed any trouble. Of course, there is no doubt that such a condition as tularemia of the conjunctiva exists, but no one could say that Parinaud's conjunctivitis is due to the bacillus of tularemia, because one can find leptothrix in the tissues in cases of Parinaud's conjunctivitis.

GLAUCOMA AND NAEVUS FLAMMEUS

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The association of glaucoma with facial nevus, accompanied in some instances by symptoms of intracranial disturbance of the opposite side, is a syndrome which was recognized many years ago but which has received little attention on account of its extreme rarity. In a rather complete survey of the available literature reports of fifty-five cases, over a period of seventy-two years, were found.

Schirmer,[1] in 1860, was the first to record the association of telangiectasis of the face and body with infantile glaucoma and enlargement of the eyeball. In addition, varicosities of the retinal veins were present.

Allen Sturge,[2] in 1879, presented a case of unilateral "port wine" mark of the face, complicated by enlargement of the corresponding eyeball; there were also changes in the choroid and tortuous retinal vessels. Epileptic seizures, on the side opposite the nevus, were attributed to a similar nevoid condition of the brain.

In 1883 Horrocks [3] reported a case of nevus of one side of the face, with involvement of the lids; the lesion was accompanied by enlargement of the globe on the same side, dilated conjunctival vessels and tortuous retinal veins. There were epileptiform attacks involving the opposite side.

W. Jennings Milles,[4] in 1883, reported his observations in a boy 15 years of age who had a vascular nevus involving the lids of the right side, and who had been blind in the right eye for nine years. Microscopic examination revealed a cavernous angioma of the choroid with retinal detachment.

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1. Schirmer, R.: Ein Fall von Telangiektasie, Arch. f. Ophth. **7**:119, 1860.
2. Sturge, A.: Tr. Clin Soc., London **12**:162, 1879.
3. Horrocks, P.: A Case of Facial and Ocular Naevus, Tr. Ophth. Soc. U. Kingdom **3**:106, 1883.
4. Milles, W. J.: Naevus of the Right Temporal and Orbital Region; Naevus of the Chorioid and Detachment of the Retina in the Right Eye, Tr. Ophth. Soc. U. Kingdom **4**:168, 1884.

Lawford,[5] in 1885, described a case of capillary nevus of the skin with an associated angioma of the choroid. Although there was no enlargement of the globe, secondary glaucoma with extensive excavation of the disk and cavernous angioma of the choroid were present.

Simeon Snell [6] wrote, in 1886, of a case of cutaneous nevus accompanied by ciliary staphyloma and tortuous vessels. Increased tension led to enucleation, and histologic examination revealed a choroid thickened by numerous vascular channels, with detachment of the retina.

Galezowski's [7] case, reported in 1898, was that of a 9 month old child with discoloration of the face which increased with the slightest cry or effort. Unilateral buphthalmos was present; however, the fundus was normal except in a circumscribed area, in which there appeared to be a choroidal nevus.

In 1902 Beltman [8] presented in detail a case of telangiectasis of the face, mucous membranes of the mouth, pharynx, nose and eyes. Both corneas were enlarged, and there was bilateral progressive glaucoma simplex. In each fundus changes were present in the pigment epithelium.

Harvey Cushing,[9] in 1906, reported three cases of unilateral vascular facial nevus, in each of which buphthalmos was present on the corresponding side. Each case also gave evidence of vascular changes in the meninges. Cushing believed that facial nevi tend to conform in outline with the distribution of the fifth cranial nerve, and that they may be associated with a hypertrophy of the deeper structures of the face, enlargement of the eyeball and a corresponding nevus of the dura.

In 1908 P. Stoewer [10] described a case of unilateral nevus of the face, associated with enlargement of the globe on the same side. Histologic examination of the eyeball disclosed angioma of the choroid.

The case of Cuperus,[11] reported in 1909, exhibited a vascular nevus of the skin of the face, angioma of the right lower lid and buphthalmos of the right eye.

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5. Lawford, J. B.: Naevus of Left Side of Face; Naevus of Chorioid, Subretinal Hemorrhage, and Detached Retina in Left Eye, Tr. Ophth. Soc. U. Kingdom **5**:136, 1885.

6. Snell, S.: Naevus Involving Eye, Brit. M. J. **2**:68, 1886.

7. Galezowski, M.: Naevus congénital sous forme de couperose occupant la face, le front et le cuir chevelu, avec buphtalmie monoculaire, Ann. de dermat. et syph. **9**:245, 1898.

8. Beltman, J.: Ueber angeborene Telangiektasien des Auges als Ursache von Glaucoma simplex, Arch. f. Ophth. **59**:502, 1904.

9. Cushing, H.: Cases of Spontaneous Intracranial Hemorrhage Associated with Trigeminal Nevi, J. A. M. A. **47**:178 (July 21) 1906.

10. Stoewer, P.: Ein Fall von Angiom der Aderhaut, Klin. Monatsbl. f. Augenh. **46**:323, 1908.

11. Cuperus: Telangiectasie des Gesichts mit Glaucoma simplex, Nederl. ophth. Gesellsch., Dec. 20, 1908; abstr., Klin. Monatsbl. f. Augenh. **47**:334, 1909.

In 1918, Elschnig [12] published the report of a case of unilateral hydrophthalmos and naevus flammeus. There was a network of dilated episcleral vessels, which were not considered secondary to the glaucoma, but which were believed to be a part of the same process as the vascular nevus.

Paton and Collins,[13] in 1919, reported a case of large unilateral nevus of the left cheek and lateral margin of the orbit, with angioma of the choroid and partial detachment of the retina. One year after ignipuncture acute glaucoma occurred.

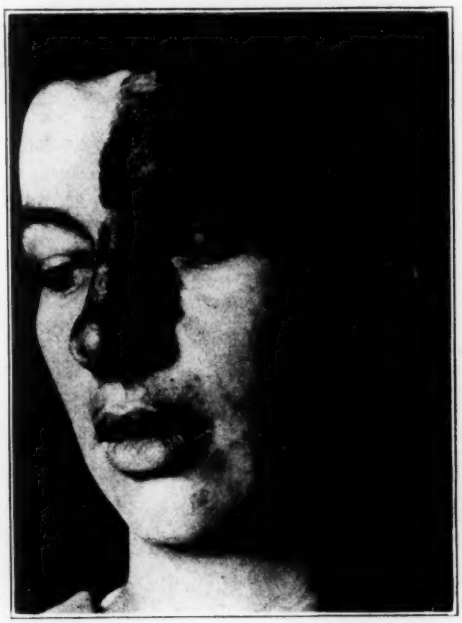

Fig. 1.—Naevus flammeus of the left side of the face.

In 1920 Freese [14] presented a man, 26 years of age, with unilateral facial nevus and enlargement of the eyeball.

In 1922 Nakamura [15] described a case of unilateral nevus with hydrophthalmos on the same side; there was an accompanying thicken-

12. Elschnig, A.: Naevus vasculosus mit gleichseitigem Hydrophthalmus, Ztschr. f. Augenh. **39**:189, 1918.

13. Paton, L., and Collins, E. T.: Angioma of the Chorioid, Tr. Ophth. Soc. U. Kingdom **39**:157, 1919.

14. Freese: Hydrophthalmus beim Erwachsenen, Verhandl. d. Berl. augenärztl. Gesellsch., Nov. 25, 1920; abstr., Klin. Monatsbl. f. Augenh. **65**:922, 1920.

15. Nakamura, B.: Angeborener halbseitiger Naevus flammeus mit Hydrophthalmus und Knochenverdickung derselben Seite, Klin. Monatsbl. f. Augenh. **69**: 312, 1922.

ing of the bones of the skull, along with those of the entire right side of the body. The iris on the affected side was poor in stroma, and radiating vessels were present over a part of its surface. Nakamura believed that nevus of the face led to hydrophthalmos, and that the same factors causing it caused anomalies of the iris and anterior chamber.

During the same year Komoto [16] described what was claimed to be the first case of facial angioma associated with glaucoma to be reported in Japan. There was a typical angioma of the right side of the face, the vessels of the bulbar conjunctiva were dilated, and glaucoma was present on the affected side.

Salus,[17] in 1923, presented two cases of unilateral facial nevus, with involvement of the conjunctiva and lids. The disk on the same side was excavated. In his cases he showed that glaucoma with nevus may not be congenital, but that it may come on later in life.

Karl Safar,[18] in the same year, contributed a case of facial nevus involving both sides of the face and the mucous membrane of the mouth. In one eye the disk was pale but not deeply excavated; in the other it was normal. Histologic examination revealed changes in the angle of the anterior chamber.

Duschnitz,[19] in 1923, described an 11 year old patient with right-sided naevus flammeus and glaucoma. The nevus involved both lids on the right side; the globe was not enlarged, but there was total glaucomatous excavation of the disk.

Löwenstein [20] reported a case of congenital naevus flammeus of the left cheek, in a woman 40 years of age, with a partial glaucomatous excavation of the right disk, but with normal tension, field and vision.

In 1924 Zaun [21] observed a spasmophilic child with naevus flammeus and congenital hydrophthalmos of the right side. The cornea, which was stippled and opaque, became increasingly cloudy, with reddening and congestion of the nevus.

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16. Komoto: Glaukom bei einer Frau mit Angioma faciei dextra, Nippon Gankakai Zashi, Dec., 1921; abstr., Klin. Monatsbl. f. Augenh. **69**:158, 1922.

17. Salus, R.: Glaukom und Feuermal, Klin. Monatsbl. f. Augenh. **71**:305, 1923.

18. Safar, K.: Histologischer Beitrag zur Frage des ursächlichen Zusammenhanges zwischen Hydrophthalmus congenitus und Naevus flammeus, Ztschr. f. Augenh. **51**:301, 1923.

19. Duschnitz: Rechtseitiger Naevus flammeus mit Glaukom, Ophth. Gesellsch. in Wien, Sitzungsb. v. Feb. 19, 1923; abstr., Ztschr. f. Augenh. **50**:246, 1923.

20. Löwenstein, A.: Aussprache, Deutsche ophth. Gesellsch. i. d. Tschechoslawakei, April 14, 1923, Prague; abstr., Klin. Monatsbl. f. Augenh. **70**:540, 1923.

21. Zaun, W.: Ueber die Beziehungen zwischen Naevus flammeus und angeborenem Glaukom, Klin. Monatsbl. f. Augenh. **72**:57, 1924.

Kiranoff's [22] case, reported in 1925, was that of a soldier, 22 years of age, with a left-sided nevus present since childhood and a blind left eye. The left side of the face was prominent, and there were exostoses over the left orbital rim. The left disk showed a glaucomatous excavation with atrophy. Kiranoff did not think it reasonable to assume that this was a case of glaucoma simplex, for after twenty-two years it appeared only on the left side. He thought that there was a definite association between nevus and congenital glaucoma.

Marchesani,[23] in 1925, recorded a case of naevus flammeus of the entire face, in a 4 month old child. Both eyelids were involved, and both corneas were enlarged. The tension in each eye was decidedly elevated, and in the right eye there was a glaucomatous excavation of

Fig. 2.—Glaucoma with deep pathologic cupping of the left nerve head.

the disk; no other changes in the fundus were observed. He was inclined to regard the association of hydrophthalmos and nevus as accidental.

In the same year Bär [24] described a case of bilateral nevus of the face, involving the lids and conjunctiva in each eye. There was no hydrophthalmos, but the temporal half of the right disk was pale, and more than half of the nerve head was included in an excavation; there were many anastomotic veins surrounding the macula. The left eye was normal.

22. Kiranoff: Zur Literatur über "Glaukom und Naevus flammeus," Klin. Monatsbl. f. Augenh. **74**:502, 1925.

23. Marchesani: Naevus flammeus und Hydrophthalmus congenitus, Wien. med. Wchnschr. **75**:2538, 1925.

24. Bär, C.: Ein bemerkenswerter Fall von Feuermal und Glaukom, Ztschr. f. Augenh. **57**:628, 1925.

In 1926 Ginzburg [25] reported a case of nevus of the right side of the face, involving the skin of the lids, conjunctiva and episclera of the right eye. Heterochromia iridis was present, the disk was excavated, and there was a coloboma of the choroid extending downward from the nerve head.

T. Yamanaka,[26] in 1927, reported a case of unilateral nevus, with glaucoma appearing in later life.

Vita's [27] case, published first in 1925 and later in 1927, showed a unilateral facial nevus over the distribution of the first and second branches of the right trigeminus, with small red spots over surface areas supplied by the eighth to eleventh spinal nerves and sacral plexus. In the right eye, hydrophthalmos was present, there was bluish discoloration of the sclera near the limbus, and the iris and fundus were darker than in the left eye.

In 1928 Knapp [28] described an 11 year old boy with a nevus involving the skin of the face, upper extremities, trunk, gums and hard palate. Bilateral glaucoma existed, with deep cupping and atrophy of the nerve heads. The irides were discolored and contained grayish nodules, and dilated vessels were present. Microscopic sections suggested angiomatous changes in the iris similar to those found in the nevus; the author suggested that these changes extending to the periphery of the iris may have caused obstruction of the filtration angle.

Kaiser,[29] in the same year, observed an 8 month old boy with nevus of the right side of the face and body. The right eye was enlarged and the tension was elevated. The author believed that true angioma of the face is not associated with hydrophthalmos, but that all cases in association are naevus flammeus.

Voegele,[30] in 1928, reported a case of right-sided nevus of the face, with low grade buphthalmos of the corresponding eye. There were increased tension and a deep glaucomatous excavation of the disk, but the vision was good and the field was normal.

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25. Ginzburg, J.: Glaukom und Feuermal mit Akromegalie, Klin. Monatsbl. f. Augenh. **76**:393, 1926.

26. Yamanaka, T.: Naevus flammeus mit gleichseitigem Glaukom, Klin. Monatsbl. f. Augenh. **78**:372, 1927.

27. Vita, A.: Idroftalmo e nevo vascolore congenito, Atti Soc. ital. di oftal., 1925; abstr., Zentralbl. f. d. ges. Ophth. **17**:555, 1927.

28. Knapp, A.: Glaucoma in Generalized Vascular Naevus of the Skin: Report of Case with Angiomatous Changes in the Iris, Arch. Ophth. **57**:219 (May) 1928.

29. Kaiser, J. H.: Einseitiger Hydrophthalmus und Feuermal derselben Gesichtshälfte, Deutsche med. Wchnschr. **54**:996, 1928.

30. Voegele: Glaukom und Naevus flammeus, Vereinigung mittel-deutscher Augenärzte, June, 1928; abstr., Klin. Monatsbl. f. Augenh. **81**:393, 1928.

Clausen [31] spoke of a case of unilateral nevus, with buphthalmos in the corresponding eye, and deep glaucomatous excavation of the nerve head in the other. Pathologic changes were not found in the chamber angle or Schlemm's canal in either eye.

Fig. 3.—Roentgenogram showing calcification of the meningeal vessels.

De Haas,[32] also in 1928, found angioma simplex of the right half of the face, associated with a blind right eye in which the tension was

31. Clausen, in discussion of Voegele.[30]

32. de Haas, H. L.: Glaukom bei Naevus flammeus, Versamml. d. nederl. Gesellsch. f. Augenh., Leiden; Nederl. tijdschr. v. geneesk. **2**:4326, 1928; abstr., Zentralbl. f. d. ges. Ophth. **20**:847, 1929.

elevated. Histologic examination showed the choroid to be from three to four times its normal thickness as a result of thick-walled and thin-walled vessels of varying caliber. This vascular anomaly extended to some of the orbital structures.

Krause,[33] in 1929, reviewed the literature and presented two additional cases. His first case, in addition to nevus of the skin and conjunctiva, showed varicose vessels of the iris, with pigment clumps on its anterior surface. The tension was increased, and there was pathologic cupping of the nerve head; the retinal vessels were tortuous and those of the choroid were greatly dilated. Compression of the carotid artery and jugular vein, with massage of the globe, resulted in reduction of tension. The second case, in addition to left-sided hydrophthalmos and angiomatosis of the skin of the face and body, exhibited convulsions of the right arm and leg and enlargement of the liver. Hemangiomas of the meninges and liver were probably present. The author thus showed the two types of glaucoma which have been mentioned in connection with facial nevus, namely, glaucoma simplex in the first case and congenital hydrophthalmos in the second.

Aynsley [34] added four cases, all of which were in female imbeciles. Each had facial nevus with enlargement of the eye on the affected side; the iris stroma of each pathologic eye was dense, giving the iris a "sculptured" appearance. Hemiplegia was present in all four cases, in three of which roentgenograms of the skull showed shadows interpreted as calcified vessels in a meningeal nevus.

Weber [35] observed a woman, 28 years of age, with a "port wine" stain of the face and body, in whom unilateral buphthalmos was present, and in whom spastic hemiplegia had existed since infancy. In the affected eye heterochromia iridis was present and the disk was atrophic. Roentgenograms of the skull suggested diffuse angiectatic changes over the left half of the cerebrum. Histologic examination of the enucleated eye failed to reveal retinal or other angiomatous formations.

Rötth's [36] case was that of a man, aged 42, with a left-sided nevus and hydrophthalmos. The vessels of the bulbar conjunctiva and the ciliary arteries were widely dilated. In addition, spastic paralysis and epileptiform convulsions had existed since birth. Roentgenograms of

33. Krause, K.: Naevus flammeus und Glaukom, Ztschr. f. Augenh. **68**:244, 1929.

34. Aynsley, T. R.: Buphthalmos and Naevus, Brit. J. Ophth. **13**:612, 1929.

35. Weber, F. P.: Notes on Association of Extensive Hemangiomatous Naevus of Skin with Cerebral (Meningeal) Hemangioma, Especially Cases of Facial Vascular Naevus with Contralateral Hemiplegia, Proc. Roy. Soc. Med. (Sect. Neurol.) **22**:25, 1929.

36. Rötth, A.: Muttermal und Glaukom, Szemészet **61**:21, 1928; abstr., Zentralbl. f. d. ges. Ophth. **20**:588, 1929.

the skull showed calcified arteries. It was assumed that the calcified vessels were those of a meningeal growth and that this led to right-sided hemiparesis and homonymous hemianopia.

Cabannes,[37] in a case of unilateral buphthalmos with hypertrophy of the structures of the face on the same side, found no evidence of any angiomatous condition in either the skin or the eye, but in the temporal fossa there was a large angioma.

Hudelo [38] observed unilateral glaucoma in a man 20 years of age with facial nevus, hemiatrophy of one side of the body and hemiparesis of the opposite side. Roentgenograms of the skull revealed an intracranial angioma.

McRae [39] reported a capillary nevus of the face and scattered nevi of the arms and hands in a girl of 14 years who was blind in the right eye. The right hand and arm were noticeably longer than the left. With the exception of the optic nerve head on the right side, which contained a goblet-shaped cavity larger than the disk itself, the eyes were normal. There was no increase in tension. Because of the association of other congenital anomalies in this case and in view of the appearance of the disk and the absence of increased tension, the author believed the condition one of pseudoglaucoma.

Suglian [40] and Zuereva [41] each added a case of naevus flammeus with associated glaucoma, and Horay [42] mentioned having seen four cases. In Zuereva's case the glaucoma did not appear until the patient was 25 years of age; in the other cases the glaucoma was of the infantile type.

In 1930 A. J. Ballantyne [43] reviewed the literature and added one case. He found, in a 3 week old infant, a unilateral facial nevus which appeared soon after birth and which followed the distribution of the trigeminus. There was an associated hydropthalmos of the left eye, with increased tension. Slight asymmetry of the facial bones was observed, the left superior alveolar ridge being more prominent than the right.

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37. Cabannes, C.: La buphthalmie congénitale dans ses rapports avec l'hémihypertrophie de la face, Arch. d'opht. **29**:368, 1909.

38. Hudelo, A.: Glaucome et naevus facial, Ann. d'ocul. **166**:889, 1929.

39. McRae, A.: Pseudoglaucoma, Brit. J. Ophth. **13**:63, 1929.

40. Suglian: Naevus flammeus und Glaucoma simplex, Oto-neuro-ophth. Gesellsch. in Zagreb; abstr., Klin. Monatsbl. f. Augenh. **83**:825, 1929.

41. Zuereva, V.: Ein Fall von Naevus vasculosus und Glaukom, Augenklin. II, Staatsuniv. Moskau, Truly II, Moskovsk. Uni. 1, 1927, p. 159; abstr., Zentralbl. f. d. ges. Ophth. **20**:847, 1929.

42. Horay, G.: Klinische Mitteilungen, Univ. Augenklin., Budapest, Szemészet **61**:45, 1928; abstr., Zentralbl. f. d. ges. Ophth. **20**:793, 1929.

43. Ballantyne, A. J.: Buphthalmos with Facial Naevus and Allied Conditions, Brit. J. Ophth. **14**:481, 1930.

Jahnke,[44] by adding the histologic findings, gave a complete report of his case, that of a 43 year old patient with naevus flammeus of the left side of the face. The conjunctival and episcleral vessels of the left globe were dilated and the cornea was enlarged. Absolute glaucoma developed. Roentgenograms of the skull showed a network of tortuous diploic veins. Histologically the chamber angle was obliterated by connective tissue, the choroid contained a typical cavernous angioma, and there was total excavation of the nerve head.

In 1931 H. T. Pi [45] reviewed the literature and added a case. The patient, a woman of 26 years, had a facial nevus involving the left side of the head, the lower right side of the face and the neck; the left cornea was enlarged and the iris was spongy and exhibited surface nodules. Compression of the carotid artery lowered the tension temporarily in the left eye.

Recently Tyson [46] has reported a case of naevus flammeus of the face and globe, associated with glaucoma, vascular changes in the iris and a calcified vascular growth in the left occipital lobe of the brain.[47]

REPORT OF A CASE

History.—C. G. M., a white woman, aged 30, entered the department of neurology of the University Hospital on April 13, 1932, complaining of blindness of the left eye, convulsions, attacks of unconsciousness and heart trouble. The patient was married and had four children, who are living and well. There was no family history of epilepsy or other nervous diseases.

The patient had been blind in the left eye since birth. At the age of 12 years, coincident with the onset of menstruation, she began to have convulsive seizures, followed by periods of unconsciousness. At first she had from eight to twelve attacks each year, but these increased in frequency until she had an average of three attacks each month.

In 1929 she noticed "palpitation of the heart." In January, 1932, she became nervous and short of breath, and had occasional swelling of the ankles. On January 21 there was an attack of unconsciousness lasting fourteen days; following the return to consciousness she had a daily convulsion for three days.

Physical Examination.—The patient was a white woman, well developed, well nourished, 65 inches (165.1 cm.) in height, and weighing 129 pounds (58.5 Kg.). The head showed no gross abnormalities. The teeth were carious. Examination

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44. Jahnke, W.: Histologischer Befund bei Glaukom und gleichseitigem Naevus flammeus faciei, Ztschr. f. Augenh. **73**:165, 1931.

45. Pi, H. T.: Hydrophthalmus in Relation to Naevus, Nat. M. J. China **17**:95, 1931.

46. Tyson, H. H.: Nevus Flammeus of the Face and Globe, Arch. Ophth. **8**:365 (Sept.) 1932.

47. Since this paper was written, the report of a case of facial nevus with infantile glaucoma has been published by Aristide Kostoulas (Naevus facial avec glaucome infantile, Ann. d'ocul. **169**:341, 1932). He believes that nevus and glaucoma are examples of congenital anomalies which are frequently associated with congenital syphilis.

of the neck revealed no adenopathy or enlargement of the thyroid. The chest was well developed and respiratory excursions normal; auscultation revealed no abnormalities. The patient had endocarditis, with a double mitral murmur at the apex, but no cardiac enlargement. There was extreme relaxation of the rectus abdominus muscle. The extremities were normally developed; no edema was present and reflexes were normal.

During the period of hospitalization the patient had three epileptic seizures, one of which was a major attack, and two of an abortive major character. During the major attack the patient was unconscious and cyanotic and there were generalized convulsions; the head was turned to the right and the pupils were fixed. In the abortive seizures the patient showed mental confusion and cyanosis, the head was turned to the right, the pupils were fixed and there was a tremor of the right arm. Neurologic examination yielded otherwise negative results.

Laboratory Examination.—The blood contained 4,610,000 red cells, 9,250 white cells and 90 per cent hemoglobin. The Wassermann reactions of the blood and spinal fluid were negative. Roentgenograms of the skull revealed evidence of irregular and somewhat scattered calcification throughout the occipital region. The calcium appeared to be in vessel walls and probably was situated in a hemiangioma of the meninges.

Ocular Examination.—In the right eye vision was 6/6. The cornea measured 11 by 12 mm. The iris was grayish blue, with distinct markings, such as are seen when little stroma pigment is present. The pupil was round and reacted to light and in accommodation-convergence. Tension was 15 (Schiötz); the exophthalmometer reading, 18 mm. Slit-lamp and ophthalmoscopic examinations revealed no pathologic changes.

In the left eye vision amounted to bare perception of light in the extreme temporal field. The skin of the entire upper lid was involved in a port wine discoloration which extended from within the hair line of the scalp downward over the entire forehead and temporal region of the left side. The left side of the nose was involved. The lower lid was normal. The skin of the affected area was thickened, and scars marked repeated attempts to remove the nevus. The caruncle was red, the nevus extending into this area; the palpebral conjunctiva of both lids was unduly red. The episcleral and deeper vessels of the globe were dilated, especially in an area situated below the lower limbus and concentric with it. There is one enormous superficial vessel in the inferior temporal quadrant, extending from the fornix to a point approximately 2 mm. from the limbus. The cornea was enlarged, measuring 12 by 13 mm. The pupil was 5.5 mm. in diameter and round, and reacted sluggishly and incompletely to light and in accommodation-convergence. Tension was 12 (Schiötz); the exophthalmometer reading, 19.5 mm. The conjunctival vessels were apparently normal; however, slit-lamp examination showed dilated, wormlike episcleral and deep vessels. There was no dilatation of the vessels of the iris; however, this structure was darker in color than that of the right eye, the alteration in color being due to an increase in stroma pigment content. The iris did not appear atrophic, but looked as if it was strewn with pigment.

With the ophthalmoscope the media appeared clear. The nerve head was slightly oval vertically and quite white; there was a deep pathologic cup which extended practically to the disk margin and which was depressed between 6 and 7 diopters, with the vessels pushed to the nasal side. The general fundus appeared quite evenly pigmented, there being no signs of choroidal vessels except in the extreme periphery. Extending upward from the temporal periphery there was evidence of an old chorioretinal disturbance, probably inflammatory, with pigmenta-

tion of the choroid and retina over a fairly diffuse area. In this region some of the choroidal vessels appeared enlarged. The retinal arteries were reduced in size; both sets of vessels showed definite white streaks and there was some overlying pigment. It was not truly typical of an old chorioretinitis; neither did it have the appearance of a nevus. The macular reflex was absent; otherwise this region appeared normal.

The diagnosis was unilateral hydrophthalmos with homolateral naevus flammeus of the face, epilepsy, mitral insufficiency and probable meningeal hemangioma.

COMMENT

Etiology.—Apparently glaucoma is associated with facial nevus only in those cases in which the lids and perhaps other ocular structures are involved. Only nine eyeballs have been examined histologically, and the reports as to the probable cause of increased tension vary. Milles,[4] Snell,[6] Stoewer [10] and de Haas [32] reported angioma of the choroid; Safar [18] found the scleral spur displaced backward, Schlemm's canal narrowed and back of its normal position, and the chamber angle covered by a web of connective tissue; Knapp [28] found angiomatous changes in the iris similar to those in the nevus. Jahnke observed cavernous angioma of the choroid and obliteration of the chamber angle by connective tissue; on the other hand, Clausen [31] and Weber [35] found neither angioma nor obstruction of the angle of the anterior chamber. In the two cases of Lawford [5] and Paton and Collins [13] a diagnosis of angioma of the choroid was made with the ophthalmoscope. Galezowski [7] suspected the presence of a choroidal nevus, and in our own case there appeared to be present choroidal nevus. Nakamura,[15] Krause [33] and Tyson [46] reported dilated vessels in the iris, and other authors have found changes in the iris stroma. Any or all of these irides may have been the seat of nevoid changes.

Many theories have been advanced to account for the associated glaucoma. Beltman [8] formulated the hypothesis that thin-walled veins in the choroid, in which the blood pressure was abnormally elevated, allowed the transudation of excessive amounts of fluid, with a subsequent increase in intra-ocular pressure; furthermore, he thought that the absorption of aqueous was diminished. Elschnig,[12] followed by Vita [27] and Clausen,[31] accounted for glaucoma on the basis of a plethoric condition of the choroidal vessels, a theory which Elschnig felt was supported by the fact that compression of the carotid artery lowered the intra-ocular pressure. Freese [14] and Rötth [36] thought that destruction of the sympathetic nerve supply was the etiologic factor. Zaun [21] felt that blood stasis increased congestion of the nevus and that this in turn led to glaucoma. Bär [24] stated the probable cause as telangiectatic changes in the choroid, and perhaps ciliary body, with increased blood pressure and slowing of the blood stream. Ginzburg [25] had the

idea that there was a congenital predisposition to glaucoma, and that vascular changes in the globe destroyed the balance between production and outflow of aqueous. Yamanaka [26] was of the opinion that there were two causes, namely, an increase in number and dilatation of the choroidal vessels, and malformation of the chamber angle meshwork with insufficient outflow of aqueous. Aynsley [34] assumed that toxins or trauma to the branchial clefts caused the formation of a cerebral nevus, which in turn affected the mesoblast of the developing eye, leading to telangectatic changes in the choroid and ciliary body with imperfect formation of Schlemm's canal or the filtration angle or to imperfect balance of the intra-ocular fluids. Cabannes [37] believed the buphthalmos to be the result of congenital hypertrophy, brought on by excessive nourishment of the tissues from the vessels of an angioma in the vicinity of the eyeball. Hudelo [38] viewed the glaucoma as a result of venous stasis caused by impairment of outflow of blood from the cavernous sinus. Tyson's [46] conception was that the increased tension was produced by interference with the excretion of intra-ocular fluids, as a result of chemical changes in the fluids from increased permeability of the vessels and also from blocking of the anterior chamber angle.

It is therefore evident that the cause is not known. However, it is reasonable to assume that nevoid, angiomatous or vascular changes in the eye or adnexa cause stasis of blood with a resultant increase in intra-ocular pressure. If, as Cushing [9] and some others believe, the facial nevus has a tendency to follow the distribution of the fifth cranial nerve, it may be that some of the intra-ocular structures supplied by this nerve are affected in a manner similar to that of the face.

Diagnosis.—Glaucoma is to be expected in cases of facial nevus which involve the lids, conjunctiva, episclera, iris, choroid or other ocular structures. As may be seen from the résumé of reported cases, glaucoma is most frequently of the infantile type; with a total of fifty-six cases it is evident that in thirty-eight the globe was more or less enlarged, a condition which does not often occur in adults. This is not surprising, since naevus flammeus is a congenital state.

That the glaucoma is unilateral as a rule is also manifest; only four cases were reported as bilateral, and in three of these the nevus was present over both sides of the face. In seven instances the nevus was bilateral, but in four of these glaucoma was present in only one eye. In one case of unilateral nevus bilateral glaucoma was present.

Changes in the iris were noted in fourteen cases: a dense stroma was reported in five cases, heterochromia iridis in five and dilated vessels in four, and once the notation was made that the iris was poor in stroma.

Changes in the choroid were mentioned in four cases; in two an ophthalmoscopic diagnosis of angioma was made, in one it was stated that a choroidal nevus was present, and in our own case a nevoid condition of the choroid was probably present.

Other ocular findings included dilated conjunctival vessels in seven cases, extension of the nevus to the conjunctiva and episclera in five instances, tortuous retinal vessels in three cases and changes in the pigment epithelium in two cases; varicosities of the retinal vessels and ciliary staphyloma were each mentioned once.

That a nevus or an angioma of the meninges was frequently present is an established fact. Cushing found vascular changes in the meninges in three cases at autopsy. Roentgenograms revealed calcified meningeal vessels in four cases, angiectatic changes in one, angioma in one, probably hemiangioma in one, tortuous diploic veins in another and enlarged bones in two. That intracranial changes were present in other instances may be surmised from the fact that in eight cases there was a hemiplegia of the body on the side opposite the facial nevus, and that in six cases there was a diagnosis of epilepsy with convulsions on the side opposite the nevus of the face. Homonymous hemianopia was observed twice.

SUMMARY

A résumé of the literature on glaucoma associated with naevus flammeus is presented, and an account of one additional case is added to the fifty-five previously reported over a period of seventy-two years. In the case herein reported there were: naevus flammeus of the face and upper eyelid over the area of distribution of the ophthalmic division of the left trigeminus; heterochromia iridis, the iris on the affected side being more heavily pigmented; a probable choroidal nevus, and a slightly enlarged blind eye, showing deep glaucomatous cupping of the nerve head but without an increase in intra-ocular tension at the time of the last examination, i. e., an arrested hydrophthalmos.

TUMORS AFFECTING THE OPTIC CHIASM AND OPTIC TRACTS

A BRIEF CRITICAL SURVEY OF THEIR CLINICAL AND ANATOMIC FEATURES

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NEW YORK

There are several diseases of the central nervous system in which visual disturbances are frequent. Chief among them are epidemic encephalitis, multiple sclerosis, various forms of syphilis of the nervous system and, above all, intracranial tumors. Some form of visual disturbance such as diplopia or blurred vision may be the earliest symptom complained of by the patient, and for this reason the ophthalmologist is often the first to be consulted in the belief that the visual defect is due to some isolated and focal disease of the eye.

Fortunately, the well informed ophthalmologist of today is fully prepared to sense a situation in which the disruption of the visual mechanism is but a local expression of a circumscribed, single or widely disseminated lesion in the central nervous system. This, of course, holds particularly true for intracranial tumors. In such cases he may be the first to recognize early papilledema, that is, an alteration in the eye-grounds, which is concurrent with and commonly corresponds in degree to the increase in intracranial tension, and is a sign highly significant and exceedingly reliable in the diagnosis of an expanding lesion. But while it serves as a secure guide for the identification of the expanding character of the lesion, it is, nevertheless, inadequate for the precise determination of the seat of the tumor. Indeed, in some instances, the *tempo* with which the papilledema develops and the *degree* to which it advances may often throw some light on the approximate location of the tumor. It is also true that, when added to other localizing signs, it leads not infrequently to a fairly accurate localization of the tumor. This is the case when the tumor is situated at a crucial point, particularly in areas where it is most likely to interfere with the flow and proper drainage of the ventricular fluid. In such event it is likely to cause a rapidly advancing hydrocephalus and accordingly will also provide a

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From the Laboratories and the Neurologic Service, Mount Sinai Hospital.

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high degree of papilledema. Hence, with tumors of the posterior fossa, arising in the cerebellum, or in the region of the quadrigeminate plate or else somewhat more forward in the interpeduncular space in the third ventricle, *papilledema will appear early, advance rapidly and reach a degree in excess of that* which is usually encountered in lesions of a still more anterior location.

However, helpful as the information obtained from the study of the eyegrounds may be, the results obtained from the study of the acuity and the fields of vision in instances in which the tumor has encroached on some part of the optic system are of a still more appreciable value. Disturbances in acuity of vision and, particularly, in the fields of vision are common in tumor of the brain and are, perhaps, more frequent than is commonly recognized. Unfortunately there seems to be still some laxity as to the frequency and the precision with which visual fields are studied and scotomas outlined. Thus, useful information is often neglected.

Reference to the diagrams depicting the optic pathways will disclose that this compact and well delineated system traverses the entire length of the brain (fig. 1). Because of its extent and its passage through crowded parts of the brain, it is exposed to frequent encroachment by expanding lesions (fig. 2). Obviously, tumors growing in the proximity of this system will result in frequent disturbances of the visual mechanism. Moreover, the course and arrangement of the fibers in this system (figs. 2, 3 and 4) are such that an interruption in the system at any given point disrupts the visual apparatus in a way peculiar and corresponding to the involved part. Thus, tumors affecting the optic nerve give rise to visual impairment different from that occurring with tumors encroaching on the optic chiasm. Again, tumors in the latter situation present an array of variable visual defects in accord with the extent of chiasmal involvement, while tumors of the temporal lobe with their distinguishing quadrantic defects and similar lesions of the occipital lobe with the more complete hemianopic fields are characterized by visual disturbances distinct from those caused by tumors encroaching more directly on the optic nerves and chiasm. Though it is quite patent that a tumor may interrupt the optic system at any point of its course, it is equally true that there are several crucial points (fig. 2) at which such an interruption will give rise to fairly typical disturbances in vision. These alone, or, more particularly, when correlated with signs arising from involvement of contiguous parts of the brain, may take the form of a series of symptom complexes. These may be designated in accordance with the relationship of the lesion to some critical point in the course of the optic pathways as syndromes and grouped as follows: (1) prechiasmal; (2) chiasmal, further subclassified as: (*a*) suprasellar and (*b*) intrasellar; (3) the temporal lobe syndrome; (4) the occi-

pital lobe syndrome, and (5) the quadrigeminal plate syndrome. In the production of such syndromes, tumors play a most important rôle. These tumors, aside from varying in location, differ also in their histologic character, to which but brief reference will be made in the discussion of each syndrome. The syndromes will be considered in the order

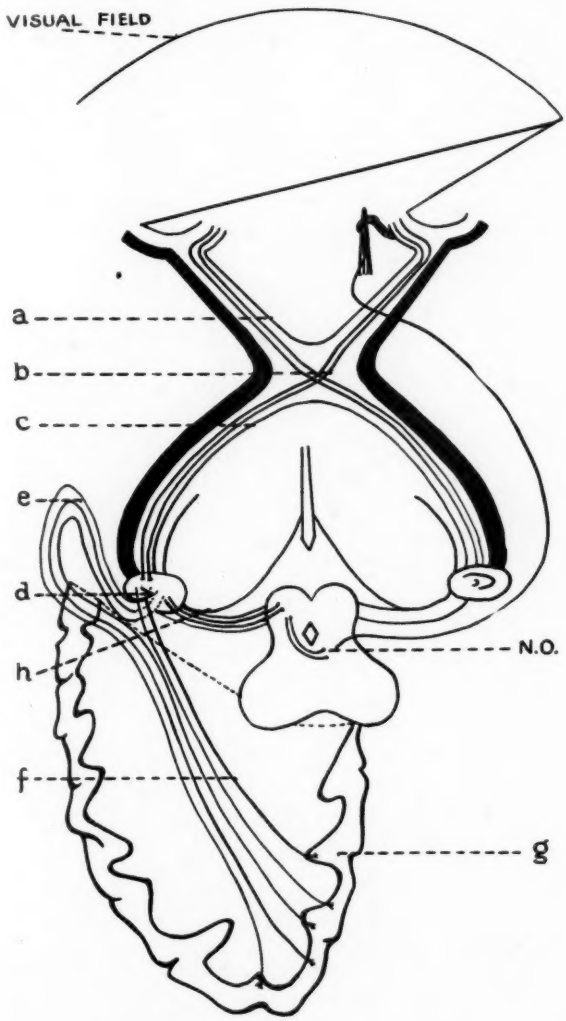

Fig. 1.—Diagram, illustrating the course and arrangement of fibers in the optic system: *a*, prechiasmal segment; *b*, chiasmal segment with the decussation of fibers arising in the nasal halves of the retina; *c*, postchiasmal segment of the optic tract, containing temporal fibers from the ipsilateral and nasal fibers from the contralateral half of the retina; *d*, the external lateral geniculate body; *e*, Meyer's loop or the "detour' of the ventral part of the optic radiation into the temporal lobe; *f*, optic radiation; *g*, calcarine zone; *h*, geniculoquadrigeminate fibers (anterior brachium); *N.C.*, nucleus of the oculomotor nerve.

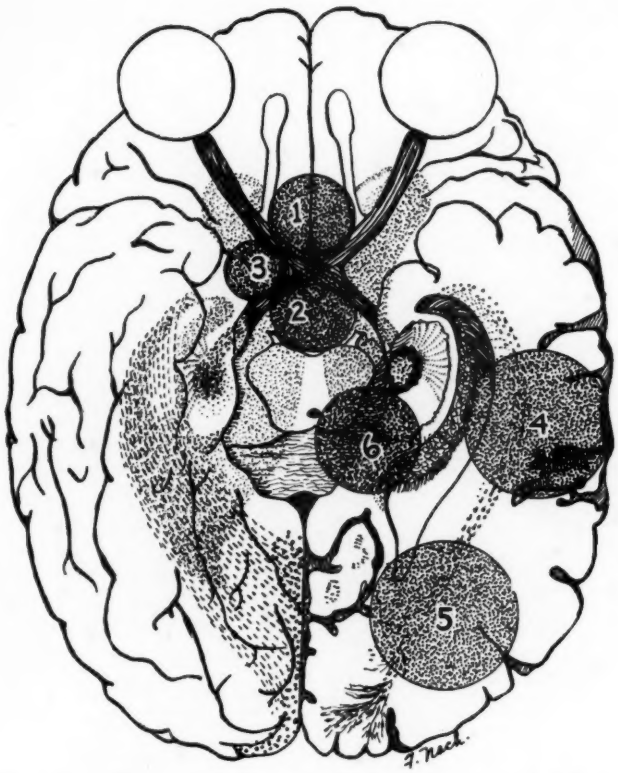

Fig. 2.—Drawing, illustrating the optic pathways and the critical points which are common sites of tumors of the brain: *1* and *2*, chiasmal tumors; *3*, tumors affecting the optic tracts; *4*, tumors of the temporal lobe; *5*, tumors of the occipital lobe; *6*, tumors of the quadrigeminal plate.

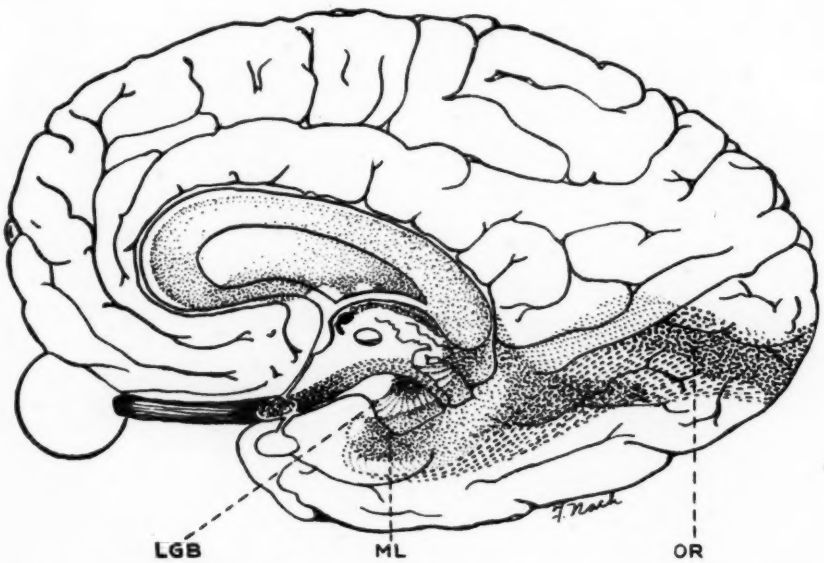

Fig. 3.—Drawing, illustrating Meyer's loop, visualized from the mesial aspect of a hemisphere (after Cushing). Note how the fibers curve about the mesial and ventral aspect of the temporal (descending) horn of the lateral ventricle: *LGB*, lateral geniculate body; *ML*, Meyer's loop; *OR*, optic radiation.

already indicated. However, since the prechiasmal syndrome belongs more properly to the topic of tumors of the optic nerves, it will be omitted and the chiasmal syndrome will be the first to be considered.

CHIASMAL SYNDROME

Under this heading several varieties will be considered, each being determined by the anatomic character of the tumor and its topographic relationship to the optic chiasm.

(*A*) *Primary Gliomas of the Optic Chiasm.*—Only tumors restricted to the chiasm or, at least, those that arise there may be properly considered in this category. This type of tumor is exceedingly rare; in my

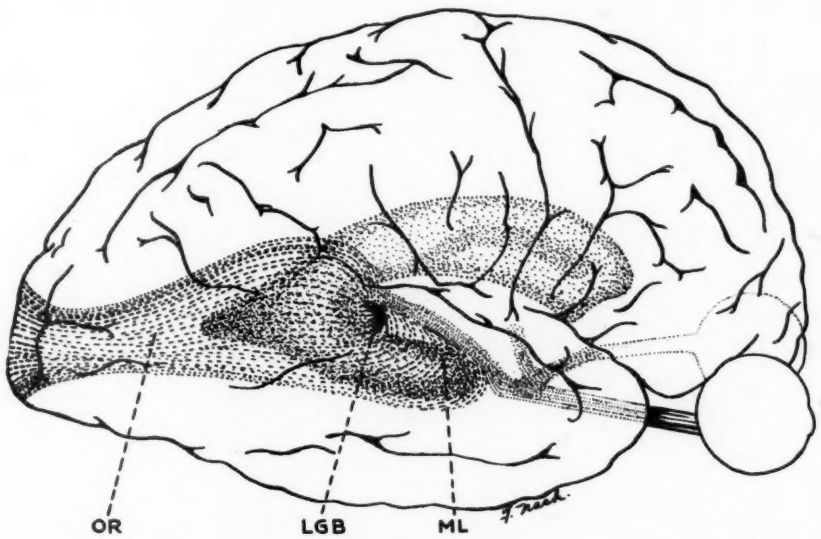

Fig. 4.—Drawing, illustrating Meyer's loop and the posterior part of the optic radiation viewed from a lateral aspect: *OR,* optic radiation; *LGB,* lateral geniculate body; *ML,* Meyer's loop. In describing the geniculocalcarine bundle, Meyer, in his original report, says: "The ventral part of the optic radiation plunges first forward from the external geniculate body into the empty temporal lobe and then backward to the calcarine cortex as the external sagittal marrow or inferior longitudinal fasciculus, which is not an association bundle, but essentially the ventral part of the geniculo-calcarine path, since there is no temporal cortex to connect with. The lateral bundle is most dorsal, from the external geniculate body to the point where it turns the posterior end of the occipital horn of the lateral ventricle to reach the calcarine cortex. The most ventral bundles are those which make the detour toward the temporal pole and end in the anterior part of the calcarine cortex. The fact that the individual bundles do not change in size on the way from the geniculate bodies speaks against giving off of fibers in their transition to occipital regions. The dorso-lateral bundle is more direct—the ventral bundle participates in the detour. The separation of fibres and the formation of Meyer's loop explain fully why tumors in the temporal lobe affecting these fibers cause quadrantic defects in the fields of vision."

collection at the Mount Sinai Hospital, there is not a single example of such a lesion, and Cushing,[1] with extensive material, has been able to record but eight cases up to date. Moreover, when the surgical and pathologic reports of the eight cases of Cushing are studied, the fact is disclosed that they cannot be properly labeled as primary chiasmal neoplasms, as in every instance a contiguous part of one or both optic nerves is involved. Under such circumstances their primary origin is highly uncertain. However, tumors which, though not true chiasmal, occur at the transition of the optic nerves into the chiasm may give rise to a set of characteristic signs and symptoms which point to the probability of a chiasmal seat of the tumor. On a rare occasion a so-called primary chiasmal glioma may thus be distinguished from tumors similarly situated but which arise from other structures in the proximity of the chiasm.

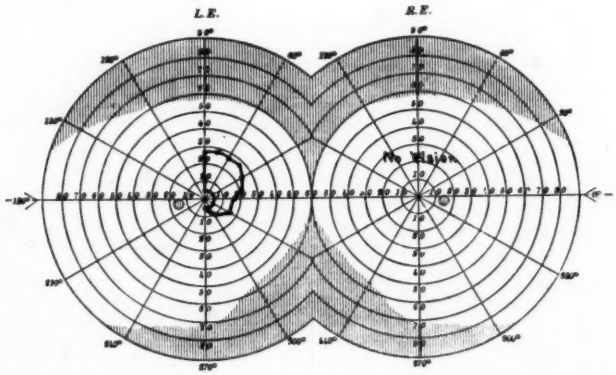

Fig. 5.—Visual fields in Cushing's case 2. Total blindness in the right eye, temporal defects in the left eye. (After Cushing.)

For lack of personal material, I shall borrow from the group presented by Cushing [1] (case 2) for purposes of illustration.

CASE 1.—A man, aged 25, suddenly noted depreciation of vision in his right eye. Soon afterward, vision in his left eye became impaired. Within three weeks there was total blindness in the right eye and further reduction of vision in the left. There was no headache, nausea or vomiting. The positive neurologic findings were: (1) bilateral primary optic atrophy; (2) total blindness with fixation of the pupil in the right eye; (3) temporal defect in the visual field on the left side (fig. 5), with vision reduced to 10/200 in the left eye; (4) a normal sella turcia; (5) no deposits of calcium in the region of the interpeduncular space.

The preoperative diagnosis was tumor of the suprasellar craniopharyngeal duct.

The operative findings were: definite enlargement of the right optic nerve at its junction with the chiasm and a suggestive increase in the circumference of the left optic nerve (fig. 6).

1. Martin, Paul, and Cushing, Harvey: Primary Gliomas of the Chiasm and Optic Nerves in Their Intracranial Portions, Arch. Ophth. **52**:200, 1923.

The patient made an apparently good recovery, with some improvement in vision of the left eye. However, six months later he became totally blind, and with the advance of some signs of intracranial disease he died six months later.

Experience with a group of seven similar cases led Cushing to conclude that: 1. The general region in which the tumor is situated may be recognized with the aid of the ophthalmologic findings. 2. The type of tumor may be distinguished in some instances from other lesions in the same location by: (*a*) the absence of changes in the sella turcica; (*b*) the absence of deposits of calcium in the suprasellar region; (*c*) the presence in some instances of other manifestations of von Recklinghausen's disease; (*d*) the early appearance of primary optic atrophy with occasional superimposition of papilledema; (*e*) the rapid progressive loss of vision associated with an unilateral temporal defect (*f*) and a peculiar lateral outline of the anterior part of the sella, which, when studied stereoscopically, gives the impression of a bulge under the anterior clinoids, though it is really due to enlarged optic foramina. In a differential diagnosis, distinction has to be made mainly between these

Fig. 6.—Appearance of the optic chiasm and optic nerves in Cushing's case 2. Note the swelling of the right optic nerve. (After Cushing.)

so-called primary gliomas of the chiasm and tumors of the craniopharyngeal duct, which have a similar location. Surgical intervention in instances of primary chiasmal glioma so far has not been crowned with success, as removal is almost impossible without serious injury to the optic nerves.

(*B*) *Tumor of the Craniopharyngeal Duct.*—This is another form of suprasellar tumor which symtomatologically is closely related to the type already discussed. I have in mind the so-called tumor of the craniopharyngeal duct. A detailed discussion of the histogenetic factors underlying the development of such a neoplasm is somewhat out of place here. Hence, I shall state but briefly some of the established facts concerning them. These tumors are teratoid in character. It has already been suggested (Globus,[2] 1923) that because of their bigeminal derivation they may be regarded as autochthonous teratomas. Their source

2. Globus, J. H.: Teratoid Cyst of the Hypophysis, Arch. Neurol. & Psychiat. **9**:417 (April) 1923.

of origin is in embryonal rests along the craniopharyngeal duct. They most commonly develop along the suprasellar course of the duct, though occasionally they may form in the depth of the sella and then, on breaking through the diaphragm of the sella, invade the parachiasmal region. This variety of tumor is the more common form of the suprasellar neoplasm.

The typical case presents a characteristic array of signs and symptoms which, in common with ophthalmologic and roentgenologic findings, make the diagnosis rather simple.

The symptoms appear most often at a preadolescent age. Because of the effect of the tumor on the hypothalamic region, they include manifestations of vegetative dysfunction such as polydipsia, polyuria, sexual and skeletal infantilism, varying degrees of adiposity and hypersomnia. These, when added to bitemporal hemianopia, involvement of the oculomotor nerves and deposits of calcium in the suprasellar region without deformity of the sella turcica, point definitely to the diagnosis of a tumor of the craniopharyngeal duct in the interpeduncular space.

It must be realized that not a small number of tumors of this type, particularly in the adult, deviate from the classic syndrome. There are those which have extended beyond their usual limits and have encroached on adjacent parts of the brain. As such they may provoke a new series of signs and symptoms which overshadow the typical signs and bring confusion into the entire clinical picture. Deposits of calcium may not be revealed in the roentgenogram; the tumors may cause defects of the visual field which deviate from the usual bitemporal hemianopia, and which often assume the character of homonymous field distortions. The following case will demonstrate such an instance.

Case 2.—A boy, aged 10, who was known to have had for some time a diffuse pallor of his disks, suddenly began to complain of headache. He vomited several times. Soon afterward he became lethargic. His condition became alarming, and he was taken to the hospital. Examination revealed: bilateral optic atrophy, limitation of gaze to the left, a moderate hemiparesis on the right, pyramidal tract signs on the right, tremor of both hands and a right homonymous hemianopia. Because of the sudden onset and rapid evolution of the picture, an inflammatory lesion in the form of encephalitis or an abscess of the brain was considered. While under observation, the patient had several attacks in which he manifested the picture of decerebrate rigidity with residual spasticity of the right side of the body. This, in view of the homonymous field defect on the right, was assumed to place the lesion in the left hemisphere. There were other findings that added to the confusion. Among them was the pleocytosis in the cerebrospinal fluid. This however, is not out of keeping with craniopharyngeal neoplasm, particularly the cystic type, as an increase in cells in the cerebrospinal fluid in such tumors is not uncommon (Beckmann and Kubie [3]). X-ray pictures

3. Beckmann, J. W., and Kubie, L. S.: A Clinical Study of Twenty-One Cases of Tumours of the Hypophyseal Stalk, Brain **52**:127, 1929.

of the skull showed no abnormality. There was a rapid decline, with the appearance of papilledema and bilateral ptosis. The patient died suddenly during one of the attacks of decerebrate rigidity, twelve weeks after the onset of symptoms.

Necropsy disclosed a neoplasm of the craniopharyngeal duct (fig. 7) residing in a widened sella turcica, and spreading upward into the third ventricle. The optic nerves were flattened.

Fig. 7 (case 2).—*A*, gross appearance of the tumor of the craniopharyngeal duct. *B*, histologic appearance of the tumor, showing small areas of calcification which were obviously responsible for the suprasellar shadows in the roentgenogram.

Many other instances deviating from the typical picture can be recited, particularly when they occur in the adult, but the last case served to emphasize the great variability in the clinical manifestations of tumors of the craniopharyngeal duct. This dissimilarity in the clinical

features is directly due to the great variation in the gross structure of such tumors and in the direction they take in their growth. Some of them are solid; others contain cystic parts, while in a fair number of cases they are thin-walled cysts. Some tumors remain limited to the interpeduncular space, while others may expand so as to implicate a variety of adjacent structures. It is the small, thin-walled cyst that offers the best promise for operative intervention. Evacuation with partial or complete removal of the cystic wall has often given brilliant results in the hands of the capable surgeon. Patients with solid craniopharyngiomas are highly unfavorable operative risks, and surgical intervention promises little in the way of restoration of normal vision.

(*C*) *Suprasellar Meningioma.*—This is another not infrequent parachiasmal lesion, which at times is clinically indistinguishable from the previously described forms. Cushing [4] was the first to call attention to its peculiarities. To paraphrase him: It is characterized by a syndrome which occurs in middle-aged patients free from endocrine disturbances and consists of primary optic atrophy, bitemporal hemianopia or a tendency in that direction, and an unaffected sella turcica, with an occasional calcium speckling in the suprasellar region. In the experience of Cushing and others, of all tumors implicating the chiasm, the suprasellar meningiomas are most favorable for operation. However, again a warning is needed. So long as the tumor, growing slowly, does not pass beyond certain limits, it may conform with all the features of the syndrome, but often the tumor remains silent, while it is still small, and is detected only when it suddenly extends beyond its usual boundaries and, by displacing contiguous structures, provokes a series of signs which deviate from the typical syndrome. This is illustrated by the following case.

Case 3.—P. R., a married woman, aged 44, suddenly noted visual impairment which on study was recognized as a left temporal heminanopic defect. With this there appeared headache; bilateral optic atrophy, greater on the left than on the right; facial weakness on the right, and visual acuity in the right eye of 8/20, and in the left eye of 8/20. A diagnosis of suprasellar neoplasm was made. X-ray pictures revealed a normal sella turcica, but widened vessel grooves, mainly anteriorly. The patient refused operation. Vision continued to decline; it was lost completely in the left eye and was markedly reduced in the right. Headaches increased. She passed into coma and was brought to the hospital. This time she showed total blindness in the left eye and, right temporal hemianopia (fig. 8). On roentgen examination, a small calcified area was visualized in the proximity of the olfactory groove. Again she refused operation. She was readmitted for the third time in coma; there was a rapid decline, with death.

Necropsy disclosed a large neoplasm in the interpeduncular space (fig. 9*A*). It extended from the anterior border of the pons to about 4 cm. from a point at

4. Cushing, Harvey, and Eisenhardt, Louise: Meningiomas Arising from the Tuberculum Sellae, Arch. Ophth. **1**:1 (Jan.) 1929.

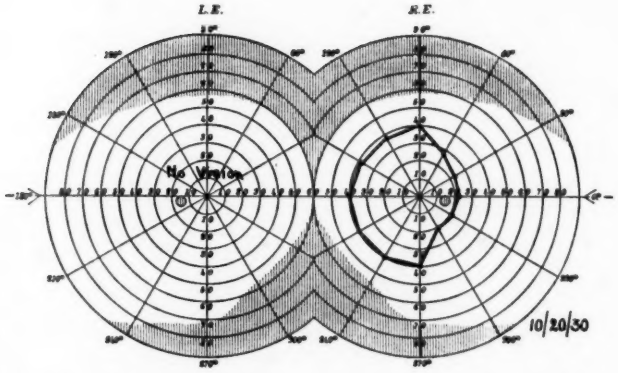

Fig. 8 (case 3).—Visual fields; test object 2 mm. white; vision of right eye, 10/40. Left eye shows total blindness; right eye a temporal defect.

Fig. 9 (case 3).—*A*, gross appearance and location of the suprasellar meningioma. *B*, histologic appearance of the tumor.

the frontal poles. It was flanked by the temporal lobes. The optic nerves, optic chiasm and tracts were hidden by the tumor. A considerable length of the olfactory tracts was also covered and compressed by the tumor. The infundibular stalk was displaced posteriorly. Histologically, the tumor was a hemangioendothelioma (fig. 9*B*).

(*D*) *Pituitary Adenoma* (*Suprasellar*).—The classic syndrome of hypophyseal tumors is too well known to be considered here at length, but attention may be called to the frequent deviations from the characteristic clinical picture which are not as fully appreciated. Thus the demand for a deformed sella turcica as essential for the diagnosis of a hypophyseal tumor must occasionally be abandoned, as there are pituitary adenomas which, because of their suprasellar location, do not distort the sella. They have either formed above the diaphragm of the sella arising from the hypophyseal duct in its course above the roof of the sella, or else they have taken root in the sella but broken through early into the suprasellar region without deforming the floor of the sella and often without causing alterations in the pituitary gland itself. They may be properly named suprasellar adenomas (Cushing[5]). They are most likely to occur in middle-aged persons, who, being free from hypophyseal symptoms as the gland is left unaffected, have no distortion of the sella and present bilateral optic atrophy and bitemporal hemianopia.

It is no less important to realize that pituitary neoplasms may be correctly diagnosed in the absence of classic bitemporal hemianopia, and that a homonymous hemianopic defect on the right is not inconsistent with a hypophyseal adenoma. It is not widely known that bitemporal hemianopia is but a phase in the development of field distortion incident to hypophyseal tumors (Cushing[6]). This becomes apparent when the changes in the fields are detected early and their progress is followed by a study of successive stages. It will then be observed that the initial visual disturbance may take the form of a small unilateral temporal defect. However, instead of developing into a typical bitemporal hemianopia, the tumor, by implicating one of the tracts, may cause the production of a homonymous defect. The following case illustrates this point.

CASE 4.—S. W., aged 32, a married woman, complained for a period of eleven years of intermittent headache. More recently headaches had become more constant and severe. Nausea and vomiting set in, and vision became impaired in the left eye. Examination revealed a pituitary habitus (obesity and coarse masculine

5. Cushing, Harvey: The Chiasmal Syndrome of Primary Optic Atrophy and Bitemporal Field Defects in Adults with a Normal Sella Turcica, Arch. Ophth. **3**:505 (May) 1930.

6. Walker, C. B., and Cushing, Harvey: Chiasmal Lesions with Especial Reference to Homonymous Hemianopsia with Hypophyseal Tumors, Arch. Ophth. **47**:119, 1918.

features); lethargy; exophthalmos of the left eye, and bilateral papilledema, greater on the left than on the right. The visual fields disclosed a right homonymous hemianopia (fig. 10). In spite of this atypical field, a diagnosis of pituitary neoplasm was made and verified by operation and necropsy.

The tumor was large and partly buried in the interfrontal region, disrupting the adjacent substance of the brain (fig. 11). Its base was in the large sella turcica. From that point it spread in all directions. It eroded the roof of the sphenoid and thinned the roof of the left antrum. It extended upward into the

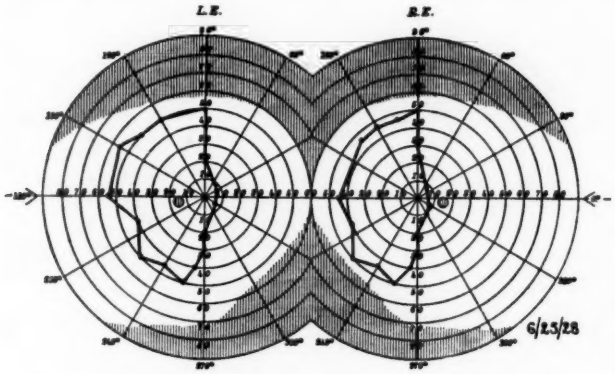

Fig. 10 (case 4).—Visual fields, revealing a right homonymous hemianopia. Test object, 5 mm. white; vision, 15/15 in both eyes.

Fig. 11 (case 4).—Gross appearance of the pituitary tumor.

third ventricle, reducing it to a narrow slit. The optic chiasm was embedded in the tumor, which histologically proved to be a pituitary adenoma.

Binasal hemianopia is a more uncommon field defect in cases of pituitary tumors. It is usually rare as an early feature. Attention on this field disturbance was recently focused by Charamis [7] and Dejean.[8]

7. Charamis, J. S.: Hémianopsie binasale par tumeur de la région hypophysaire, Ann. d'ocul. **168**:737, 1931.

8. Dejean, C.: L'hémianopsie bi-nasale, Arch. d'opht. **47**:219, 1930.

The explanation for such an occurrence has already been projected by Knapp,[9] who believed that displacement of the chiasm by a midline tumor may force its uncrossed fibers against the internal carotid arteries and thus, by indenting these fibers, causes the binasal hemianopia.

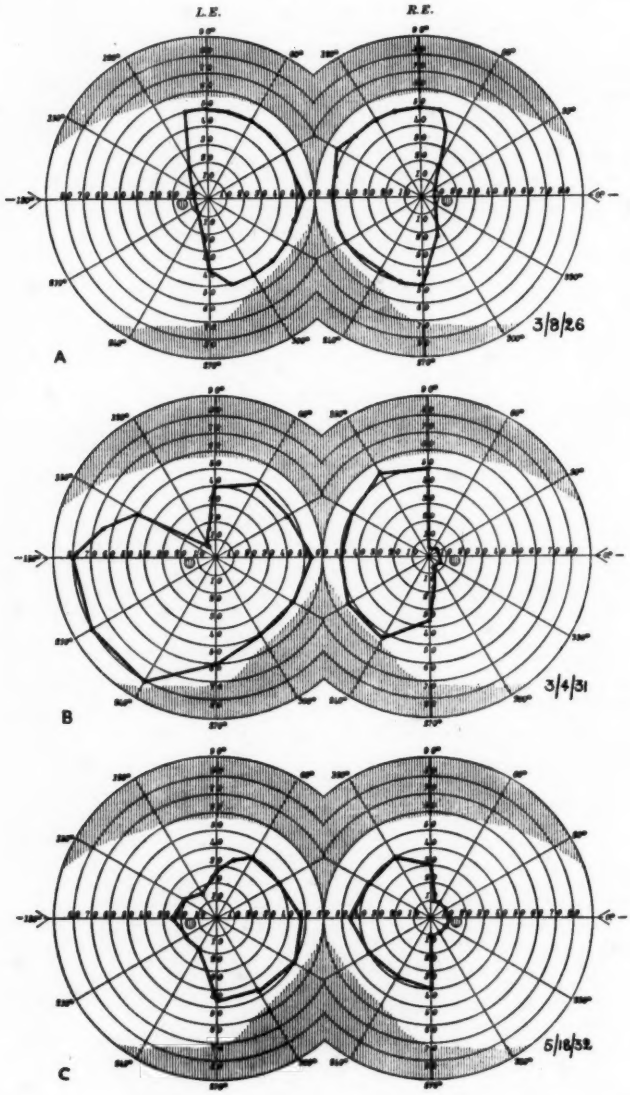

Fig. 12 (case 5).—Visual fields. Test object, 10 mm. white; vision, 20/50 in both eyes.

Surgical intervention in pituitary adenoma yields, beyond question, the most satisfactory results. It must also be borne in mind, however,

9. Knapp, H.: Hemiopic and Sector Like Defects in the Field of Vision and Their Connection with Diseases of the Heart and Brain, Arch. Scient. & Pract. Med. & Surg. **1**:293, 1873.

that if the surgical step is delayed in such tumors, they are apt to grow and expand beyond available surgical measures.

Occasionally high voltage roentgen or radium therapy may give rise to apparent improvement and to the false hope that the pathologic process has been arrested. However, in the course of time an explosive reappearance of symptoms will shatter one's belief that the disease process may remain permanently dormant. Unfortunately, at such a time the expanded lesion has extended into such regions as to make surgical approach prohibitive. This is what occurred in the following case.

CASE 5.—J. J., an unmarried woman, aged 31, gave a history of sudden cessation of menses at the age of 15, after normal menstruation for a period of two

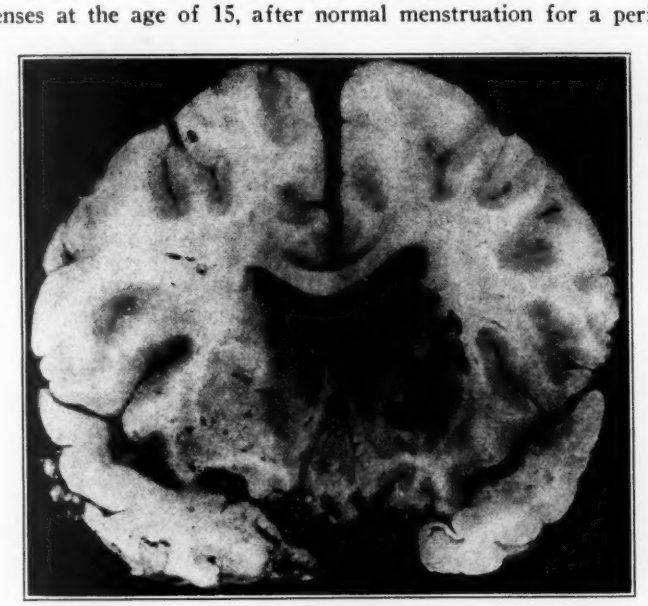

Fig. 13 (case 5).—Coronal section of the brain, showing extension of the tumor upward into the interlobar region, and also softening in the capsular region in the left hemisphere, not unlikely due to a disturbance of the blood supply to this region. This lesion accounts for the hemiplegia which suddenly developed before the patient's last admission to the hospital.

and one-half years. At the age of 21, there were a rapid gain in weight and a gradual impairment of vision. These changes were accompanied by periodic headache and vomiting. At the age of 26, a diagnosis of pituitary tumor was made. The visual fields showed fairly typical bitemporal hemianopia (fig. 12*A*). An attempt to reach the tumor by an intranasal approach failed. Radiotherapy was instituted, and gratifying improvement followed; the visual fields showed marked widening (fig. 12*B*). The condition remained stationary for five years. However, sudden development of right-sided hemihypalgesia, dysphagia, dysarthria and facial paresis brought the patient to the hospital again. There were pronounced bilateral optic atrophy, a right internal rectus paresis, deviation of the tongue to the right and pyramidal tract signs on the right. X-ray pictures of

the sella turcica showed enlargement similar to that seen on the admission five years previously. The fields showed marked constriction (fig. 12*C*). A few concretions were seen below the anterior clinoids. Craniotomy was considered imperative.

During the course of the operation respiratory embarrassment suddenly developed, and the temperature rose to 105 F., while the blood pressure dropped rapidly. The patient died before the dura was opened.

Necropsy showed the pituitary body as a small rounded protrusion, but in the interpeduncular space on the coronal section of the brain there was a large extension of the tumor dorsally, into the third ventricle (fig. 13) and adjacent parts. It is not improbable that the removal of this tumor in the early stage might have altered the sequence of events.

TEMPORAL LOBE (MEYER'S LOOP) SYNDROME

Lesions of the temporal lobe, when not accompanied by such localizing signs as uncinate seizures, visual hallucinations or typical speech disturbances, often serve as the most severe test of diagnostic ability. Not so much when they are situated in the left hemisphere, for there the temporal anomia may be a deciding factor in the localization of the lesion, but when the lesion is in the right hemisphere, the focal signs may be so meager as to offer no clue to the actual position of the lesion. In such instances a knowledge of the course and distribution of the geniculocalcarine fibers is of immense service. Cushing [10] focused attention on the so-called Meyer's loop [11] forming part of the geniculocalcarine tract. Its significance is found in the part it plays in the causation of a partial or so-called quadrantic type of hemianopic defect. It is this visual distortion that may often be the only deciding diagnostic sign to secure a correct localization in a tumor of the temporal lobe. It is of immense service in the following diagnostic dilemma.

CASE 6.—J. K., a man, aged 44, aside from an attack of unconsciousness four years previously, was well until two and one-half months prior to his admission to Mount Sinai Hospital. He then began to complain of severe headache, which occurred in protracted attacks. The headaches increased in intensity, and vomiting of the projectile type appeared. At the same time vision became defective. Examination revealed the following positive findings: (1) hypersomnia: (2) bilateral external rectus weakness, greater on the right than on the left; (3) advanced bilateral papilledema; (4) central facial paresis of the thalamic type on the left; (5) left-sided hemihypalgesia; (6) mild pyramidal tract signs on the left side, with slight weakness of the left upper extremity; (7) a Babinski sign on the right. There was no gross hemianopia. With these findings, no accurate localization was possible, aside from placing the lesion in some part of the right hemisphere. A lesion in the posterior part of the frontal or in the parietal lobe could readily be assumed in accord with the foregoing findings. It was only on

10. Cushing, Harvey: Distortions of the Visual Fields in Cases of Brain Tumor, Tr. Am. Neurol. A., 1921, p. 374.

11. Meyer, Adolph: The Connections of the Occipital Lobes and the Present Status of the Cerebral Visual Affections, Tr. A. Am. Physicians **22**:7, 1907.

careful plotting of the visual fields that the site of the tumor in the temporal lobe was established. Again, a single perimetric tracing would have been insufficient and could have easily misled; only by successive charting of the visual fields was their true character identified (fig. 14).

Necropsy revealed a tumor in the right temporal lobe. It encroached but slightly on the parietal region. Histologically, it was designated as a transitional glioma.

OCCIPITAL LOBE SYNDROME

The most characteristic sign of a tumor of the occipital lobe is the homonymous hemianopia, particularly when it is an isolated finding.

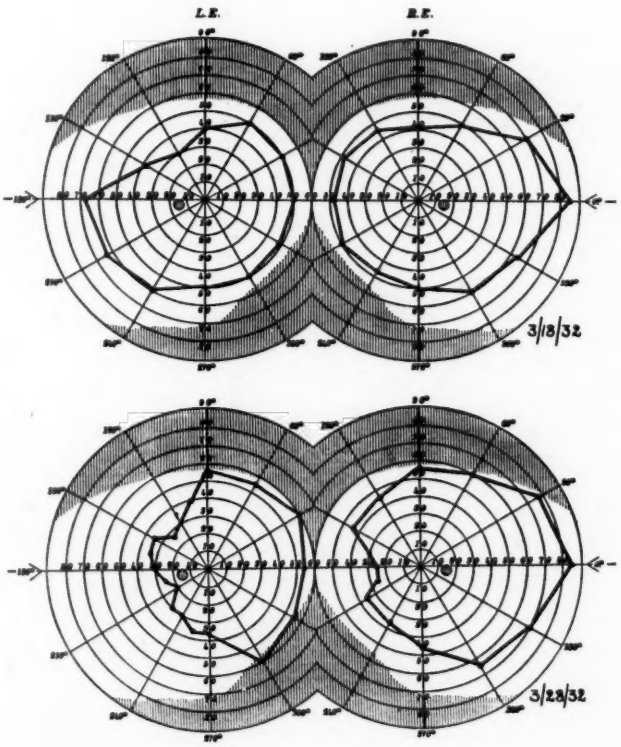

Fig. 14 (case 6).—Visual fields. Test object, 2 mm. white; vision, 20/50 in both eyes. Central color scotoma in the right eye.

Alone, however, it would be insufficient to place the lesion with certainty in this region, as homonymous visual defect, as was already shown, is a frequent finding also in tumors of the temporal lobe. The academic distinction between the negative or psychic hemianopia associated with tumors of the occipital lobe as contrasted with the positive hemianopic blindness due to a lesion of the optic tract is of little assistance, as it is difficult of practical application. In an exceptional instance an intelligent patient may recognize the loss of half vision and thereby indicate the presence of a positive hemianopia. Such aid is certainly valuable, but

it is seldom obtained. The same is true of the Wernicke hemianopic pupil in chiasmal or postchiasmal lesions. It, too, is difficult of demonstration. Of much greater significance is the character of the hemianopic fields. It was recently pointed out by Horrax and Putnam [12] that not one of the thirty patients with tumors of the occipital lobe studied had hemianopia of the quadrantic type, and that in all of them the macular bundle was preserved. This feature is, of course, of immense service in distinguishing tumors of the occipital lobe from those of the temporal lobe. The character of hallucinations may occasionally be utilized as a localizing lead. It is said that the unformed type is characteristic of tumors of the occipital lobe, while the formed visual hallucinations are more common in lesions of the temporal lobe.

In neoplasms of the left occipital lobe the problem of localization is less difficult, as there the hemianopia is often associated with optic aphasia and word blindness merging into alexia. Here, as in other postgeniculate lesions, the preservation of the pupillary reflexes is of service.

The diagnosis of a tumor of the occipital lobe may also be aided by contiguous signs, such as those which arise from implication of the thalamus and midbrain. The following case will serve well to demonstrate this feature.

Case 7.—A man, aged 41, was admitted to the Mount Sinai Hospital for the first time on Aug. 15, 1925, on the third day of an acute illness. Four and one-half months previously he had suddenly fallen and lost consciousness. He remained in bed for several days and had then apparently recovered. He vomited repeatedly on that day and several times on the following day. There soon developed intense headache, marked rigidity of the neck, generalized weakness, unsteadiness of the hands, a slow speech and a slight rise in temperature (from 100 to 101.5 F.). He revealed unequal pupils, which reacted well to light and in accommodation. The margins of the disks were indistinct. There were marked rigidity of the neck, a bilateral Kernig sign, a slight facial weakness and hyperactive deep reflexes, more marked on the right side. The pulse was slow. Lumbar puncture yielded a dark brown, apparently bloody fluid. The blood pressure was 95 systolic and 50 diastolic. The Wassermann reaction of the blood and cerebrospinal fluid was negative.

On the second day the patient suddenly had a convulsive attack. Following the attack there were noted a tremor of the head (the head was inclined to the right and the chin carried to the left), ataxia of the right upper extremity, spontaneous nystagmus to the right and past pointing inward with the left hand.

The diagnosis at this time rested between meningo-encephalitis and intracranial aneurysm. During the next two months this condition improved sufficiently to permit the patient's discharge from the hospital. The final diagnosis was aneurysm of a cerebral artery at the base of the brain, with bleeding into the subarachnoid space.

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12. Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumor: VII. The Field Defects and Hallucinations Produced by Tumors in the Occipital Lobe, Tr. Am. Neurol. A., 1932, p. 115.

The patient remained at home in apparently good condition for about six weeks, and then, two weeks prior to readmission, he began to complain again of intense headache, dizziness and impaired vision. He began to vomit, and an unsteady gait, falling to the left and weakness in the left arm and leg developed.

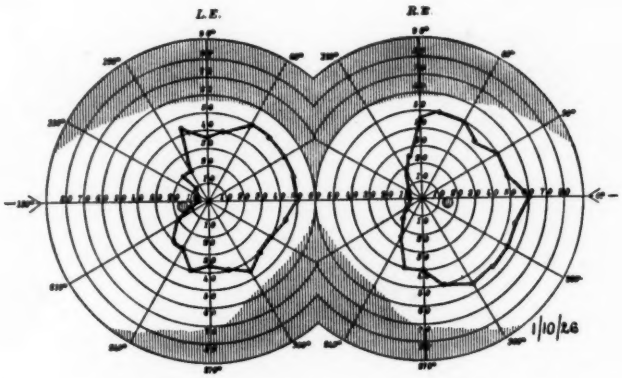

Fig. 15 (case 7).—Visual fields.

Fig. 16 (case 7).—Gross appearance and location of the tumor.

On the second admission, he showed slight clouding of the intellect; unequal pupils, the right reacting poorly to light; a few nystagmoid twitchings to the right; bilateral papilledema; left homonymous hemianopia (fig. 15); left hemiparesis; hyperactivity of the deep reflexes, the left being more lively than the right; less active abdominal reflexes on the left; a bilateral equivocal plantar reflex; ataxia in all four extremities, with cerebellar hand movements on the left side, unsteady gait, with a tendency to fall to the left and backward; a

cerebellar tilt of the head to the right, the chin being carried to the left; mild astereognosis, and slight disturbance in the joint sense on the left side. The cerebrospinal fluid was under slightly increased pressure, but was clear and contained no cells.

Course.—At this time the seat of the lesion was placed at the undersurface of the brain supratentorially in the region of the right temporo-occipital lobe. Such a localization explained most of the focal signs, such as the left homonymous hemianopia, the mild paretic phenomena on the left and some of the cerebellar features, as well as the manifestations of increased intracranial tension.

To verify the diagnosis a left ventricular puncture was carried out. The fluid was found slightly blood-tinged and under increased pressure. No other unusual observations were made. The patient was returned to the ward in a good condition. During the course of the next two days there was noted a progressive mental deterioration with further elevation of the disks. He died suddenly two days after the ventricular puncture.

Necropsy.—This revealed a large tumor on the undersurface of the right temporo-occipital region. It measured approximately 7 by 4 cm. on cross-section, and was situated mesial to the optic radiation. It extended beyond the mesial aspect of the right occipital lobe, and dislodged the mesencephalon to the opposite side, compressing the brain stem in the region of the quadrigeminate plate (fig. 16). Its histologic structure was that of a "spongioblastoma multiforme."

THE QUADRIGEMINAL PLATE SYNDROME

Objections will be raised to the inclusion of this syndrome among those arising from interference with the optic pathways. However, reference to figure 1 will convince one that the short association fibers connecting the lateral geniculate and the anterior quadrigeminate body are part of the optic system, and that their interruption may cause some disturbance in the ocular mechanism; thus, paralysis of upward gaze, skew deviation and Argyll Robertson pupils, which are characteristic of this syndrome,[13] may be traced to some disorder in this link of the optic pathways. However, what is more essential is that expanding lesions of the type described as pinealomas often grow forward into the supratentorial region (Globus[14]) and thus may implicate some part of the optic tract and give rise to hemianopic defects. In none of the pinealomas so far recorded was hemianopia described. However, it does not mean that field disturbances never occur in tumors of the pineal region. It is more likely that the failure to elicit such defects is due to poor cooperation on the part of the patient, because of either youth or impaired intellect, conditions so frequently found in patients with tumors of this type.

13. Globus, J. H.: Tumors of the Quadrigeminate Plate, Arch. Ophth. **5:** 418 (March) 1931.

14. Globus, J. H.: Pinealoma with Supratentorial Extension and Manifestations of Disrupted Temperature Regulation, Libman Anniversary Volumes, New York, International Press, 1932, vol. 2, p. 491.

CONCLUSIONS

The preceding discussion was mainly concerned with some diagnostic points aiding in the localization of intracranial tumors affecting the optic pathways. However, it would be incomplete without a few remarks as to the therapeutic measures available for the amelioration of the conditions considered. Of course, this problem belongs rightly to the realm of the neurosurgeon, who is in a better position to discuss it. However, the neurologist and the ophthalmologist, though mainly concerned with the recognition of the lesion and its localization, cannot relinquish their responsibility when the problem of surgical intervention is raised. They must aid in predicting the probable anatomic character of the tumor in question, which, though accurately localized, may often be of such a type as to preclude a successful removal, as in the case of spongioblastoma multiforme. They must be fully aware that in some instances a tumor may involve adjacent structures to such an extent as to make its removal incompatible with preservation of life, or that it may be so intimately interlaced with certain structures as to make impossible its removal without destruction of vital parts of the system. This is true of tumors in the interpeduncular space, which are likely to wrap themselves about the optic chiasm or optic nerves in such a way that removal without complete loss of vision is impossible. However, such conditions cannot always be predicated with certainty, and only by exploring the intracranial contents can the character and relationship of the lesion be verified with accuracy, as in the case of primary chiasmal gliomas and cysts of the craniopharyngeal duct.

Fortunately neurosurgery today provides a sufficient margin of safety in exploratory craniotomies. Such a step is thus fully warranted even in conditions in which the removal of the tumor is highly problematical, particularly when the patient, faced by the prospect of eventual blindness, is willing to take what is thought to be a desperate chance. It would seem, then, that once the existence of an expanding lesion in the cranial cavity is recognized and localized, it is highly advisable, if not to say imperative, to explore the region suspected. The experienced neurosurgeon may be relied on to sense an unfavorable condition, which should deter him from an attempt to remove the tumor when such a step is beyond the limits of safety or when partial or radical removal will soon be followed by recurrence, as in malignant spongioblastomas.[15] He may be trusted to realize in other instances that its removal would incur irreparable injury, as in some tumors of the craniopharyngeal duct or in primary chiasmal gliomas. He is also fully prepared to recognize

15. Globus, J. H., and Strauss, Israel: Spongioblastoma Multiforme: A Primary Malignant Form of Brain Neoplasm, Arch. Neurol. & Psychiat. **14:** 139 (Aug.) 1925.

situations in which the risk incident to the removal of the tumor is consistent with the chance for a cure. Such is the case in tumors which, when identified early, are readily removed with excellent results. Among them are the meningiomas, pituitary adenomas, benign gliomas and even a few of the tumors of the craniopharyngeal duct.

Thus, in conclusion, it may be said that the appearance of symptoms pointing to the presence of an expanding intracranial lesion demands a thorough search for signs aiding in the identification of the character of the tumor and the recognition of its site. All available diagnostic means must be employed for the accurate localization of the lesion. Among them visual fields hold a most prominent place. Detection of what on first glance appears to be an insignificant distortion of the field of vision may serve as the only lead to the site of an otherwise silent location of the tumor.

OPHTHALMOLOGY IN THE BIBLE AND IN THE TALMUD

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It is difficult to ascertain definitely the status of medicine among the ancient Hebrews. The Bible and particularly the Talmud deal primarily with laws and ethics, and in the course of discussions only do they refer to medicine as to other vital subjects of that day.

Neither the Bible nor the Talmud can be considered a medical treatise. The Mishnah [1] does mention a medical work called "Sefer-Refuoth," [2] and the Talmud quotes a treatise on pharmacology, "Megillath Sammamin," [3] but neither of these books has survived the long centuries of Jewish migration. Nothing that has been transmitted from the ancient Hebrews can parallel the work of Hippocrates,[4] of Galen [5] and of Celsus,[6] nor can one compare Jewish medicine with ancient Egyptian medicine as it is typified by the Papyrus Eber.[7] Medical subjects as found in the Talmud and Rabbinic writings are of a fragmentary character. The subject matter is far and wide apart. There can be no question, however, that while ancient Jews were

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Most of the works referred to throughout this article are in the Judea-Aramaic language. A few are in second century Hebrew. They are known as tracts or tractates. Some of the Talmudic tractates were translated into the English, German and French languages. The Radkinson translation in English is of a fragmentary character. It is defective as well as incomplete.

1. Mishnah is derived from the Hebrew "shana," to learn, to repeat, and applies to a compilation of laws by the Patriarch R. Judah, who lived about 200 A.D., a contemporary of Galen.

2. Pesachim.[56a]

3. Yoma 38a.

4. 560-470 B.C.

5. 200 A.D.

6. 25 B.C.-50 A.D.

7. This is the earliest record of ophthalmology known, dating from 1565 B.C. It is in reality a collection of prescriptions in detail. The diseases are mentioned only by name, without any description, much the same as in the medical writings of the ancient Greeks. The Papyrus enumerates thirty different ocular conditions (diseases or symptoms) for which treatment is suggested. Dr. George Eber, a German archeologist, purchased this document from an Arab, who discovered it in the year 1872 between the legs of an ancient mummy in the Necropolis of Thebeth. The Papyrus is found in the library of Leipzig.

influenced in medicine as in many other matters by the Babylonians from the north and by Egyptians and Greeks from the south, they developed their own theories and practices in the art of medicine.

Writers of the Old Testament viewed conditions of health and of disease as emanating from Divine sources. "I wound and I heal," said the Lord.[8] "I God am your healer."[9] A physician, therefore, was considered the messenger of God, whose duty was to carry out the will of the Lord.[10] Disease and death were predestined, over which the physican had no control. Penitence and prayer were the only things that could change the fate of a person if he was once marked to be sick as a punishment for sin.[11] The doctrine, "Of what has to be, cannot be changed," taught by the Stoics,[12] was later responsible for the fatalism believed in by Islam; namely, that the natural process of the world cannot be changed by man.

The doctrine of predestination, however, was not accepted by many sages of the Talmud.[13] "No one," said R. Yanai, "shall put himself to think the Lord will do miracles with me and cure me himself."[14] Rabbi Ismael[15] logically explained the Old Testament verse relating to assault, "and he shall cause him to be thoroughly cured,[16] by saying that permission is given for the physician to heal, and that he is to be paid for his services by the one who committed the assault.[17] Con-

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8. Deuteronomy 32:39.

9. Exodus 15:26.

10. Zohar, vol. 3, p. 299.

11. II Chronicles 7:13; Jona 3:10.

12. Si fatum tibi est, ex hoc morbo convalescere, sive medicum adhibueris, sive non, convalesces.

13. Talmud is the name of two large encyclopedic works, which have been preserved as the products of the Babylonian and Palestinian schools, extending from the third to the fifth centuries A.D. The name Talmud is derived from the word limud, and denotes learning.

14. Sabbath 32a.

15. Berochoth 60a. Preuss, Julius: (*a*) Biblisch-Talmudische Medizin, Berlin, S. Karger, 1911, p. 26; (*b*) p. 20; (*c*) p. 303; (*d*) pp. 302 and 303; (*e*) p. 310.

16. Exodus 21:19.

17. Berochoth 60a. The New Testament followed the same teaching; thus, one reads, "and Jesus answering, said unto them, they that are whole, need not a physician, but they that are sick." Luke 5:31. Matthew 9:12. Mark 2:17. A story is told of the same Rabbi, that one day when he and his colleague, Rabbi Akibba, were administering to the sick of Jerusalem, a peasant rebuked them saying, "Aren't you interfering with the ways of the Lord? The Lord punished them with sickness for their sins and you are trying to heal them." "Aren't you doing the same thing?" they replied. "God created the earth to provide mankind with food and you don't wait for him to plow, to fertilize, and to sow, but you do it yourself." Midrash Samuel, chap. 4.

trary, however, to the custom of other ancient people, the Hebrew priests had no jurisdiction over the art and practice of medicine.[18] To them were assigned the duties of a health officer, such as the supervision of patients suffering from diseases like leprosy and gonorrhea,[19] which were viewed by the writers of Pentateuch as highly communicable diseases.

That medicine became a highly esteemed profession among ancient Jews may be seen from the apocryphal writings of Jesus, son of Sira.[20] "Honor a physician with the honor due unto him," said he, "for the uses which ye may have of him, for the Lord had created him . . . and he has given men skill, that he might be honored in his marvelous work. . . . My son in thy sickness be not negligent. . . . Give place to the physician. . . . Let him not go from thee for thou hast need of him." [21]

According to the Talmud, physicians might charge comparatively high fees for medical services. "A physician who takes nothing is worth nothing," was a popular expression in the Talmudic period.[22] To live in a town that had no physician was considered unsafe and extremely hazardous.[23] Circuit physicians were employed by several communities where the people were too poor to pay for the services of the physician.[24]

During the last Jewish commonwealth down to the Talmudic days physicians were employed in the Israelitic Courts of Justice, whose expert testimony was received and whose opinion was final in criminal matters. In matters of assault, for example, the physician's duty was to pass on the character of the injury received by the plaintiff, and punishment was meted out to the defendant in accordance with the physician's opinion.[25] When the judge pronounced corporal punishment, it had to be under the supervision of a physician.[26]

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18. In ancient Greece, physicians were at one time called the priests of Aescalopius. Hippocrates was named an Aescalopian, but never a priest.

19. The prophets did sometimes resort to the art of medicine. Elijah brought life to a child who was at the threshold of death (I Kings 17:11, 22); his disciple Elisha performed a similar miraculous cure (II Kings 4:17, 20); another prophet restored vitality to the paralyzed arm of King Jeroboam (I Kings 13:4, 6); Isaiah is reported to have administered a cataplasm made of figs to the inflamed parts of King Hezekiah (II Kings 20:7), and Jesus restored to health patients inflicted with incurable diseases (Matthew 14:14).

20. 180 B.C.

21. Sira 38:1, 2, 3, 4.

22. Baba Kamma 85a.

23. Sanhedrin 17b.

24. Exodus Rabba 46:3.

25. Sanhedrin 78a; Gittin 12b.

26. Makoth 22b.

The local judicial council had to pass on the rights of the physician to practice in the particular locality.[27]

The knowledge of the medical man in the Talmudic days was based first on tradition,[28] second on observation of diseases and third on experiments on animals.[29]

The knowledge of anatomy possessed during Talmudic days was of the kind derived from the dissection of slaughtered animals. The dead human body was held in great respect among ancient people. To the Hebrews, the very touch, or even entering the room, of the dead necessitated isolation for seven days, followed by thorough cleansing of the person and disinfection of the room and of all things that had come in contact with the dead body.[30]

The Romans followed the old Hebrew regulations. The Pontifex-Maximus and his Augurs were prohibited to look on the dead.[31] Even Galen never dissected a human body himself, and when he saw, at the schools of Alexandria, a human skeleton, he was greatly impressed.

However, despite the fact that the Old Testament discouraged, for religious and sanitary reasons, any contact with a dead body, it did not prevent Jewish students of the second century A. D. from studying anatomy on the human cadaver whenever possible. The students of R. Ishmael, who had some doubt as to the generally accepted tradition that the number of bones in the human body was 248,[32] obtained, after much effort, the body of a young harlot who was executed under the Roman Law. They subjected the body to a boiling process in order to get rid of the tissues, and found the number of bones to be 252.[33] Of course, neither of the numbers given agrees with modern anatomic knowledge,[34] but the explanation of the differences may be found in the youthful age of the subject used.

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27. Baba Bathra 21a.

28. Hullin 93a.

29. Sanhedrin 5b.

30. Numbers 19: 11, 13, 16, 20. Ezechiel 6: 5, 9, 7. II Kings 23: 14, 16, 20.

31. Tacitus Annals, p. 62.

32. It appears that the teaching that the human body contained 248 bones was a general tradition among anatomists and physicians, as late as the twelfth century. The celebrated Arabic physicians, Avicenna and Abul-Casam, also gave the number of bones in the human skeleton as 248.

33. Bekhoroth 45a.

34. According to modern anatomists, the skeleton consists of 200 bones.

The Talmud mentions one famous Hebrew osteologist, by his Greek name, Theodorus Horophe,[35] who enjoyed a wide reputation also as a physician.[36]

The anatomy of the vital organs of the human body received careful attention. The heart is described as being composed of ventricles, the right somewhat larger than the left, and being situated to the left of the median line.[37]

Rab, the famous teacher of Babylon, expressed a view far in advance of his time; namely, that the aorta (Koneh-shel-leb) contained blood and not air.[38]

Such a view was revolutionary, for Hippocrates and his school, and even Galen, at a later date, believed the arterial system and the aorta to be air tubes; hence the names, artery and aorta.

The lungs, the liver[39] and the generative organs are minutely described, and all other organs, such as the pharynx and larynx, the esophagus, the stomach and the intestines, were carefully studied in the Talmudic academies. The medical terminology in the Judea Aramaic dialect scattered in Talmudic literature would make a good-sized medical dictionary.

Physiology as it is understood today is based, in addition to the knowledge of anatomy, on the sciences of physics and chemistry. Very little of these sciences was known in the first eight centuries A. D. Many natural phenomena today attributed to physical forces and chemical actions were "terra incognita" in the Talmudic days.

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35. Tosefta Kethuboth 4. Theodorus Horophe is sometimes known as Theodos. He probably studied in Alexandria, the only place autopsies were permitted (Preuss [15b]). Rophe is the Hebrew name for physician. The Aramaic term is Assia.

36. The opinion of Theodorus on medical matters was frequently sought by scholars in the academies of Palestine. To mention just a few cases: One time, while digging the ground, the villagers of Tamina found a pile of human bones. The bones were taken to the Academy of Lydda, to see whether they were from one person or from more. Theodorus was called in, and he decided that they were from several bodies (Tosefta Ahaloth 4, 2). Another time he was requested by the academy of Jabneh to give his view in a case in which, on examination of the organs of a slaughtered animal, it was found that the uterus was missing. R. Ishmael, the head of the Academy, taught that an animal cannot live any length of time without that productive organ and therefore its flesh could not be used for food. Theodorus, however, gave as his opinion that the absence of the womb does not endanger life and the meat may be eaten, adding that all cattle leaving Egypt had their generative organs removed so that the fine species of cattle of Egypt would not be cultivated elsewhere (Tosefta Kethuboth 4. Sanhedrin 93a).

37. Menohoth 37b. Hullin 45b. Jerusalem Talmud, Truma 8.

38. Hullin 45b. Jewish Encyclopedia, vol. 8, p. 411.

39. Hullin 46a-b; 45a; 47a; 93 b. Tamid 31a.

"The human body as all other matter in the Universe," said Rabbi Rehumai,[40] "was composed of four elements, fire, water, earth and air," a theory long before advanced by Hippocrates, the father of medicine, and the celebrated Ionic school (600 B. C.). The human body as a whole, Hippocrates stated, consisted of moisture, dryness, heat and cold, the different constituents of the human body being made up of the four elements in various proportions. In addition to the four elements there were four cardinal fluids, called humors.[40] Blood consisted of moisture and warmth, and was composed of the elements water and fire (Sanguins). Mucus was made up of moisture and cold from the chemical elements of water and air (phlegma). Yellow bile was a mixture of dryness and warmth; the elements that composed it were earth and heat (Choli). Black bile was made of dryness and cold, of the elements earth and air (Melancholi).[41]

Considering such a confused state of medical knowledge, it is interesting to find in Talmudic literature numerous quotations that are both illuminating and instructive. A few will suffice. "Life of all organs in the body depends on the heart."[42a] "Respiration is the process of burning." "Expired air cannot sustain life."[43] "Fever serves as a protection to the body against diseases."[44] "The stomach proper performs a purely mechanical function, that of churning the food. The

40. Midrash Tmura 1. Reziel Jamalach, p. 12.

41. Footnote 40. Health was defined as a condition in which there existed the proper combination and harmonious action and reaction of the various humors on each other, and on the various solids and tissues. Disease was the improper combination and unharmonious action of these humors. The natural creative forces of the body Hippocrates called Physis or nature; hence the name physician or naturalist. When Physis is left to itself, sickness normally runs a certain course through three successive stages—crudity, coction and crisis. In the first of these three stages, the fluids or humors degenerate; in the second, preparation for their removal takes place; the third stage is that of recovery. The crisis was especially wont to occur on odd days (Yalkut Genesis 34, 25. Psalms 41, 4). This is a relic of the old Pythagorean philosophy, which taught that a physician must beware of all odd days and of the following even days—the fourteenth, thirtieth and forty-second days from the beginning of the disease. See Hirschberg, J.: Geschichte Augenheilkunde bei den alten Griechen, in Saemisch, Theodor: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1899, vol. 12, chap. 23, p. 69. (The apprehension for odd days is emphasized in the Talmud, Pesachim 110b.) The brain was considered to be a large gland the function of which was to secrete the humors and to disperse them throughout the body. The humors injurious to the parts they came in contact with, or when not of the proper quality or quantity, produced diseases of the various organs.

42. (*a*) Zohar, vol. 3, p. 227; (*b*) p. 129.

43. Sanhedrin 77a.

44. Nedarim 41a.

digestion is carried on in the intestines." "The contact of food with the mucous membrane produces stimulation of the organ and opens the mouth of the lymphatics." [45] "The end of the period of digestion manifests itself by a return of the desire for food." [46] "Spitting in public places spreads diseases." [48] "During an epidemic one should keep indoors and keep away from crowds." [49] "The reason faculties are lodged in the brain." [50] (Regarding the last quotation, it should be stated that the general belief among students of the day was that the center of reasoning was the heart.)

The object of this article is to present in as brief a form as possible the art of ophthalmology known to the ancient Hebrews of the Biblical and Talmudic days, embracing a period of about twelve hundred years, and not to make a survey of medicine in general as practiced by the ancient Hebrews. Those who are interested in the subject may be referred to the admirable work "Biblisch-talmudische Medizin" by Dr. Julius Preuss.[15]

An introductory remark as to ophthalmologic terminology is necessary. It is difficult in many instances to identify the Aramaic names of ocular diseases. Many mistakes crept in while copying from one manuscript to another. Other terms lost their original meaning during the long period of two thousand years in those countries where the Aramaic language was not spoken. The terms of ocular diseases in most cases could be made out only by the description of the symptoms of the case, and where no symptoms are given, one is compelled to search for a corresponding word in one or the other cognate languages, such as: Hebrew, Arabic, Syriac, Assyrian, Ethiopic and Coptic, as well as Greek and Latin.

Attention should also be called to the fact that the Talmud is not the work of one man; it is a collection of opinions and sayings of more than three thousand scholars,[51] embracing a period of about six hundred and fifty years, on all subjects known to antiquity that were discussed in the academies of Babylon and Palestine.

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45. Reziel Hamalach 13.

46. Bekhoroth 52b. Jewish Encyclopedia, vol. 8, p. 412.

47. Deleted.

48. Derech Erez Rabba 9.

49 Baba Kamma 60b.

50. Yebomoth 9a. Midrash Mishle, chap. 1.

51. The word Rabbi, contrary to the general understanding, should not be defined as theologian, minister, clergyman or priest. Rabbi, etymologically speaking, signifies great, and is applied to a scholar, a teacher, a head of an academy or a master in a particular field of learning.

THE EYE

The eye in Hebrew is called *Ayin,* its name being derived, according to Talmudic authorities, from *Ain,* a well or a spring.[52] Ancient people looked on the eye as the well of the human body, because in that organ is situated the fountain of tears.[53] (It must be remembered that the lacrimal apparatus was not recognized by ancient anatomists.[54])

Another reason given why the eye is like a well is that that organ is made up of watery parts, such as the humors. "As a spring," remarks a Talmudic writer, "brings forth water, so the eye keeps up refilling itself, and as a well becomes useless the moment its sources dry up, so the eye, if the fluids are failing, loses the pleasures of sight.[55]

Another Talmudic authority compares the eye to an illuminating well, the reflection of which is brought about by the watery humors.[56] The author of that statement was undoubtedly influenced by the teaching of Hippocrates that the aqueous, vitreous, and crystalline humors were the center of vision.[57] The optic nerve itself, according to Hippocrates, was a hollow tube, through which the humors of the brain flowed, and caused frequently all forms of diseases, such as ophthalmia, glaucoma and cataract. Cataract signifies a waterfall, and the word is an inheritance from the early schools of medicine.

The theory of the father of medicine that the centers of vision were situated in the humors of the eye [58] was not agreed to by other Greek sages. Alcmaeon [59] and Democrates' theory of vision was that colored images were being continually detached from objects and that these little masks or casts pressing into the eye were fixed on the pupils until the soul was able to take note of them.

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52. Sotha 3, 2. Sanhedrin 108. Cited in Midrash Horephuoth, p. 13.

53. The Semitic race had a marvelous propensity to weep and to pour out a flood of tears (II Samuel 3, 32. Psalms 6, 7. Luke 19, 41. Jerusalem Talmud 9, 17. Lamentations 3, 48).

54. R. Hiyah Ben Guria knew the connection of the eye with the mouth (Nidah 55b).

55. Midrash Seichel Tob Genesis 16, 7.

56. Psikta 7, 1, 3. (In modern poetry one finds: "Thine eyes are springs in whose serene and silent waters heavens are seen." William Cullen Bryant.)

57. Midrash Mishle 1. Early writers believed that the brilliant crystalline lens situated in the center of the eye produced vision. Abul-Casam, for example, stated that "in the interior of the eye are three moist bodies. The center one is the crystalline lens from whence sight emanates. The function of the coats of the eye is to guard this precious lens" (HaMeati, Nathan: Amur ben ali, Mausuli, Hebrew edition, chap. 5).

58. Galen, many centuries later, taught that the function of the retina is to announce to the brain the changes that have been induced in the corpus crystallinum.

59. Alcmaeon is one of the earliest of the Pythagorean philosophers, who discovered the optic nerve.

In this connection it should be remembered that at one period the ancients looked on the eyes as the windows through which the soul peeped, and that the vision itself was emanating from the soul.[60] A far more advanced theory is that of Aristotle.[61] "The objects perceived," he said, "excite motion in the higher centers of the brain. In consequence the sense organs are acted on. What we see in the object is color, but this we do not see directly. We see it through the medium of light which sets the color in motion and causes production of impressions on the organs. Without the light we see no color, hence no object either."

Injuries to the brain, according to the Talmud, would cause blindness by forcing fluid from the brain to cover the eye.[62] This coincides with the Hippocratic theory, which states that the cause of all diseases is the descent from the brain of seven different kinds of injurious humors or slimes. The third humor from the brain, flowing into the eye, causes ophthalmia. Glaucoma and other disturbances are produced by the contact of the sixth humor with the ocular organ. Recovery from ophthalmia was marked when these injurious humors began to thicken and form concretions around the eye and its inflamed parts.[63]

Ancient ophthalmology was confined to diseases of the external segments of the eye and the parts of the organ visible to the naked eye. Diseases of the fundus undoubtedly did exist, but it was impossible to diagnose them before the ophthalmoscope was invented (Helmholtz, in 1845). Two large divisions of ocular diseases were recognized: ophthalmia, which included all varieties of congestive disturbances, and blindness, which embraced all cases of loss of sight, partial or complete. The latter was not considered a symptom of some disease in the media or the back of the eye, but it was the disease itself.

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60. It was a general belief among the ancients that the abode of the soul was the place of perpetual light and that the soul itself was a ray of light. To the New Zealander, not only was the eye the seat of the soul, but he also believed that when the body descends to the original clay the eye triumphs over the body by ascending to the skies, where it becomes a star in the celestial galaxy. Even medical writers of the middle ages could not free themselves from this belief. Thus, Abul-Casam, in explaining the mechanism of sight, stated that vision is brought about when the soul passes from the brain into the pupil (Amur ben A Al-Mausili, translated by J. Hirschberg, J. Mittwoch and J. Lippert, Leipzig, von Veit Company, 1908). The eye is spoken of in modern poetry as follows: "Heart in her lips and soul within her eyes." Byron. "The eye is the window of the soul, the mouth, the door; the intellect, the will, are seen in the eyes."—Hiram Powers.

61. 374-321 B. C.

62. Baba Kamma 98a. Midrash Horephuoth, pt. 2. Teil-Abib, p. 20.

63. Hirschberg, J.: Geschichte Augenheilkunde bei den alten Griechen, in Saemisch, Theodor: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1899, vol. 12, chap. 23, pp. 68 and 70.

OCULAR ANATOMY

The Talmudic knowledge of the anatomy and physiology of the eye may be briefly described as follows: The eye (*galgal-ha'ayin*) is a globular organ situated in the cavity of the orbit (*hoor-ha'ayin*), and is embedded in a mass of fat within the bony cavity of the skull, secured and protected from injury in front by the lids (*afapayim*), lashes (*risim*) and brows (*gebinim*).[64] The latter were considered an important protection to the visual organs. It was customary for ancient people to promote the growth of long hair from the eyebrows in order to prevent the sweat of the head, in tropical climates, from flowing into the eyes. A story is told about old Rabbi Johanan whose eyebrows were so long that a pin, made for the purpose of keeping the hair of the brows up, had to be used by his students in order not to obscure his vision.[65]

The eyelids are also known as *shmuros* (watchers), because of their function in common with the lashes to guard the eye against foreign bodies, wind and the glare of the sun.

The eyeball is composed of seven tunics [66] (in this description the Talmud differs from the teaching of the early masters Hippocrates and Galen, who describe only three coats), as follows: ocular conjunctiva, sclera, cornea, choroid, retina, iris and lenticular capsule.[67]

The sclera (*haloben-shebeayin*) is described as a white membrane the structure of which is formed of fatty tissue.[68]

The pupil is termed *Ishon* (*Babas-Ayin* or *Bath-Ayin*) because of the miniature image seen in the pupil when one looks into it. Ishon signifies little men, the diminutive of *Ish*. *Babas-Ayin* is the baby of the eye. The iris (*Kesheth*) is described as being situated at the inner junction of the black and white coats and is called *Sira*.[69] Back

64. Baba Kamma 38a. Nidah 23. Zohar 3: 126. Derech-Erez Zuta 9. Gebinim is from Gaboth, a bow.

65. Baba Kamma 117a. Aristotle also speaks about the long hair of the eyebrows (De Anima 3: 11, 74).

66. Tekunei Hazohr, p. 70.

67. That the eye was composed of seven tunics appears to have been a general tradition among medieval Semitic scholars, such as the celebrated physicians Avicenna (980-1037 A. D.) and Abul-Casam (996-1050 A. D.). The seventh tunic, according to the Arabic conception, is the anterior capsule of the lens.

68. Bekhoroth 38b. This is evidently Aristotelian teaching (De Anima 3: 17, 89).

69. Strange to say, the Bible has no word descriptive of the iris. In other Semitic languages cognate to Hebrew, such as Arabic, it is known as Madirian (mother of the eye). In neo-Persian it is similarly named Madar-i-ain. In Hebrew it would be Aim-Ayin, the mother of the eye that surrounds and embraces the pupil, called the daughter of the eye. The neo-Hebraic form Kesheth signifies a rainbow because of its various colors. It is also known as "comet shebeshohor" (Tikunei ha Zohar 9, 70).

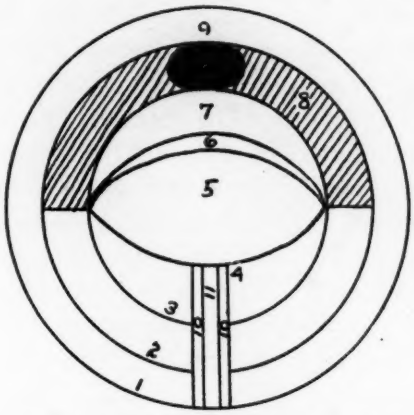

Fig. 1.—Eighth century drawing of the eyeball. *1* indicates the sclera; *2*, the choroid; *3*, the retina; *4*, the vitreous; *5*, the crystalline lens; *6*, the spider web tunic or the anterior capsule; *7*, the aqueous humor; *8*, the iris; *9*, the pupil; *10*, the optic nerve; *11*, the optic canal.

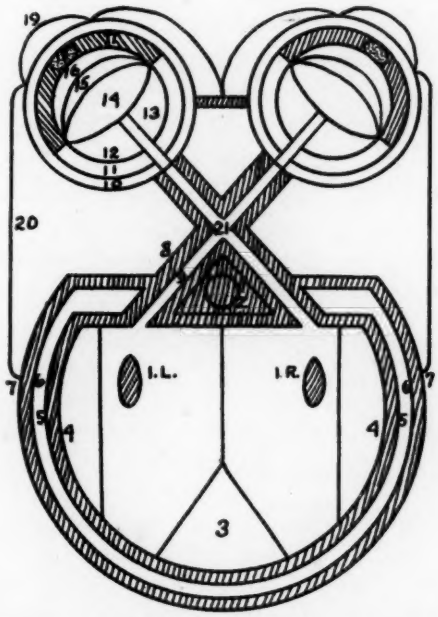

Fig. 2.—Tenth century drawing of the eye (Abul-Casam). *1L* and *R*, *2* and *3* indicate the skull cavity; *4* and *5*, the dura mater; *6* and *7*, the skull and skin covering; *8*, the optic nerve; *9*, the optic canal; *10*, the sclera; *11*, the choroid; *12*, the retina; *13*, the vitreous; *14*, the crystalline lens; *15*, the spider web tunic or the anterior capsule; *16*, the aqueous humor; *17*, the iris; *18*, the pupil; *19*, the cornea; *20*, the ocular conjunctiva; *21*, the optic chiasm.

of the pupils are the humors; in regard to their function, the Hippocratic theory is generally accepted by the authors of the Talmud.[70]

A far advanced view as to the structure of the interior of the eye, probably inspired by Aristotle, is given by the author of Zohar, a cabalistic work of the second century B. C. "In the interior of the eye," said the author of Zohar, "are found many small nerves and blood vessels. The center pillar (meaning disk) produces sight. One branch goes up to the top of the head and another branch terminates at the heart." [42b]

It appears that the aforementioned mystic writer traced the optic nerves up to the cerebral centers; there they unite to form the optic chiasm. One branch then turns left and, passing down along the left side of the trachea, enters the cardiac muscle. The part of the optic nerve that terminates in the brain is responsible for sight. By reason of its direct connection with the heart,[71] the eye is influenced by all vital organs of the body, and it follows, according to the Talmud, that any disease of the vital organs in the body will leave an imprint on the eye.[72] A diagram of the distribution of the optic nerve thus described is transmitted through a letter of the Hebrew alphabet called *Ayin,* which signifies eye [73] (fig. 3).

While general diseases in Palestine in Biblical days, thanks to its salubrious climate and the simple life of its inhabitants, were not as common as in the neighboring lands,[74] ocular diseases appear to have been of frequent occurrence among Palestine Jews.[75] The diseases, however, are not often easy to identify at the present time. Ancient writings are like archeologic relics on pottery or stone that were buried under a heavy mound for milleniums. The fragments must be pieced together and the worn out and erased symbols filled in before one is able to decipher the inscription. The recovery of facts concerning diseases of the eye buried in the large and worm-eaten volumes of ancient Hebrew literature can be accomplished only by a similar piecing together of fragments of scattered sayings by connecting the misspelled words of languages no longer spoken or written.

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70. The light emanating from the eye proceeds from the watery substance within the eye (Psikta 7, 1, 3).

71. Abodah Zara 28b. An Aristotelian theory is that all nerves of the body originate from the heart. Taanith 24a.

72. It must be borne in mind that the letters of the Hebrew alphabet are named for their resemblance to certain objects. Thus, Alpha means a branch; Beth, a house; Gimmel, a camel; Daled, a door, and Ayin, an eye.

73. Divrei-Yemei Yehoshua, cited in Ozar Israel, letter E, New York, 1910.

74. Tacitus Historiae 5, 6. Corpora humonum salubria et ferentia laborum.

75. Hashash ainayin, eye trouble, is repeatedly mentioned in Talmudic literature (Jerusalem Talmud Sabbath 11b). Jerusalem Sukah 53a.

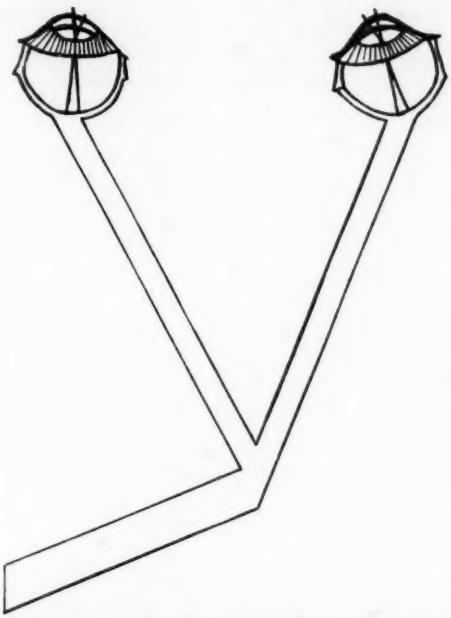

Fig. 3.—Ayin, the sixteenth letter of the Hebrew alphabet.

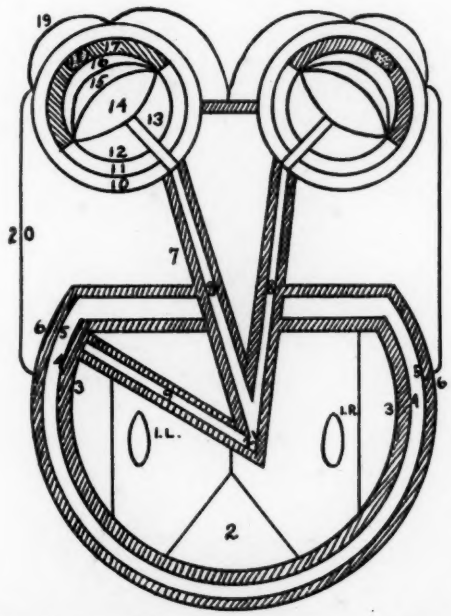

Fig. 4.—The Talmudic conception of the eye. *1L* and *R* and *2* indicate the skull cavity; *3* and *4*, the dura mater; *5* and *6*, the skull and skin covering; *7*, the optic nerve; *8*, the optic canal; *9*, the branch to the heart; *10*, the sclera; *11*, the choroid; *12*, the retina; *13*, the vitreous; *14*, the crystalline lens; *15*, the spider web tunic or the anterior capsule; *16*, the aqueous humor; *17*, the iris; *18*, the pupil; *19*, the cornea; *20*, the ocular conjunctiva; *21*, the optic chiasm.

The identification of diseases of antiquity leaves a most indelible imprint on the history of medicine, linking together the Talmudic *Rophe* with the scientific physician of today.

OCULAR PATHOLOGY

Ocular pathology of the ancient Hebrews may be divided into diseases of the lid and of the eyeball. The first embraces diseases of the lids and conjunctiva.

Jabeleth.[76]—A hard wart or cutaneous horn is a circumscribed growth, round or irregular in shape, situated beneath the bulging skin. It is also found in animals. According to the Mosaic law, when an animal is troubled with a *jabeleth* on the eye, it is not fit for sacrifice because *jabeleth* is a permanent defect.[77] Rabbi Hanina defines *jabeleth* as a growth or wart on the lids. According to the Rabbi, a *jabeleth* may grow on any part of the body. Some Talmudic writers [78] differentiate between a hairy and a hairless growth, and claim that *jabeleth* is a scleral excrescence.[79] The hairy *jabeleth* may be a dermoid cyst, which, according to Bayer, is frequently seen on the eyes of sheep. Hard and soft *jabeleth* are also described.[80]

Dildul.—The famous commentator Rashi translates *dildul* in the old French dialect *verrue-verruca*, a wart, probably a hordeolum, which is a soft growth of common occurrence situated at the margin of the lids. Jastrow,[81a] however, describes the tumor as having a thin neck, and his definition would suggest a molluscum simplex, a cutaneous growth attached by a pedicle to the skin of the lids.[82]

Chalazion (*Arabic, chilzuna; Hebrew, hilazon* [83]).—This, etymologically speaking, is a conchiferous animal, such as a snail, an oyster or a purple fish, and it is a term applied in Gemara [84] to a growth on the eye or its appendages. No description of the growth or its place is available in the Talmud. Because of its phonetic similarity to the

76. Leviticus 22:22.

77. Bekhoroth 6:10.

78. Bekhoroth 40b.

79. Tosefta Bekhoroth 6, 2.

80. Bekhoroth 40b.

81. Jastrow, Marcus: (*a*) Dictionary of the Targumim and Talmudim, letter dildul; (*b*) p. 550.

82. Fuchs, E.: Text-Book of Ophthalmology, translated by Alexander Duane, Philadelphia, J. B. Lippincott Company, 1911, p. 593. Preuss.[15c] Die Augenheilkunde des Ibn Sina, translated from the Arabic by J. Hirschberg and J. Lippert, Leipzig, von Veit Company, 1902, chap. 16.

83. Bekhoroth 38b. Tosefta 4:2.

84. Gemara is another name for the Talmud more applicable to the Babylonian system of Jewish tradition; it is the Aramaic word for study.

Greek *Chalazion* (a small hailstone), Benjamin Musaphia [85] concluded that the Aramaic term was adopted from the Greek. *Chalazion* applies to a chronic disease of the lids characterized by the enlargement of the meibomian glands and is due to stenosis of the glandular ducts. However, the tumor of the lids understood by the word *Chalazion* in no way resembles a snake. It is therefore more likely that the Aramaic *hilazon* was a pterygium, a disease of the eyeball, synonymous to the Hebrew word *nachash*. That the Aramaic term originally was considered an ocular condition described as pterygium is borne out by the statement of Rabba,[86] who, in doubt as to what *hilazon* was, made inquiries of R. Johanan, a senior physician, who in turn told him that it was the same term as *nachash*.[87]

Trichiasis (*Shachbana* [88]).—The Talmud describes this term as a condition in which the hair of the eyelashes rests on the eyeball, and if a priest suffers from such a malady he is incapacitated from attending to his priestly duties. Because of its damage to the cornea, his sight will become permanently defective.[89] The Arab physicians have taken trichiasis as the chief symptom of trachoma, and not the granulations.

Injuries to the lids [90] were considered incurable. (This agrees with the teachings of Aristotle.) A priest suffering from injuries of the lids could not attend to his priesthood,[91] and an animal having this defect was not fit for sacrifice.[91]

Blepharospasm (*Sone-shemesh*).—Etymologically, this term means "hatred of the sun," but it is applied to continuous blinking. The Talmud says that the people of Palmyra blink with their lids because they live in sandy places and the wind blows sand into their eyes.[92] A story is told of the people of Machuza, a town on the banks of the Tigris, who lived in dark houses, which caused their eyes to blink.[93]

Ptosis (*Zanahas*).—Drooping of the eyelids was attributed to old age, which caused weakness of the upper lids, as is seen from

85. Benjamin Musaphia was a Hamburg physician and lexicographer of the seventeenth century.

86. Rabba was a Talmudic physician of the latter part of the third century.

87. Baba Kamma 38b. Sanhedrin 91a. Sabbath 26a.

88. Bekhoroth 44a. Tosefta Bekhoroth 5:2.

89. Dr. Marcus Jastrow (Dictionary of the Targumim and Talmudim, letter Schachbana) and other commentators are of the opinion that Schachbana refers to the hair of the eyebrows, as it is known that the ancients permitted long hair to grow over their eyes, but such a condition could not be considered a permanent defect, as it could be removed.

90. Bekhoroth 38a.

91. Bekhoroth 44a.

92. Sabbath 31a.

93. Berohoth 59b.

Genesis (48, 10): "And the eyes of Israel were heavy for age, so that he could not see." The word "heavy" in Genesis in all probability means difficulty in lifting the upper lids. The Hebraic word *cabdu*, which literally means heavy, could not mean dim,[94] for in the verses preceding [95] and succeeding [96] that passage, it is evident that Israel could see.

The Talmud also cites a notable case of ptosis in that of R. Dossa Ben Horkenos.[97] A term for ptosis is not found in the Bible or in the Talmud. *Zanahas* is a neo-Hebraic form.

Lagophthalmos (*Tordos*).—A condition in which there is an incomplete closure of the palpebral fissure is mentioned in the Talmud.[98] A priest suffering from such a defect was unfit to attend to ecclesiastical duties. No etiology or treatment is given for that disease.[81b]

Blepharitis Ciliaris.—This appears to have been a common disease among the ancient Jews of Palestine.[99] The chief symptoms were complete or partial loss of cilia (*thamir*), redness of the margins of the lids and closure of the lids by a thick crust.[100] The etiology of blepharitis marginalis is mainly due to excessive weeping.[101] The disease itself is not mentioned in the Bible, but under the term *thamir* in the Talmud (loss of cilia), blepharitis is generally applied. *Thamir* was considered one of the early symptoms of leprosy and the cause of partial or complete blindness. That this condition of the eye was thought injurious to sight is corroborated by pseudo-Galenic writings, in which it is stated that loss of lashes interferes greatly with sight for both near and distant vision.[102] According to the Talmud, a priest was disqualified to perform priestly functions if he lost his eyelashes or if his eyelashes were broken at the root.[103] Maimonides (1131-1205), a Jewish physician of the middle ages, did not agree that the

94. As rendered in St. James' version, the Hebrew word for dim would be Cahu.

95. Genesis 48:8.

96. Genesis 48:11.

97. Yebomoth 16a. Cited in Midrash Horephuoth, p. 20.

98. Bekhoroth 44a. Midrash Horphuo, vol. 2, p. 18.

99. A notable case is that of Leah, the daughter of Laban. According to the Old Testament narrative (Genesis 29:17), "Leah had weak eyes, but Rachel was beautiful in appearance." The weak eyes of Leah evidently spoiled her appearance. She evidently had redness and swelling of the margins of the lids and loss of eyelashes with the formation of concretions on the margins. Her case could not have been one of tender eyes as rendered in St. James' version, for such eyes could hardly be called ugly and give cause for complaint on the part of Jacob.

100. Mikvaoth 9:2-4. Nidah 67a.

101. Baba Bathra 123a.

102. Kings 14:102.

103. Bekhoroth 7:3. Sanhedrin 23b.

loss of cilia hindered vision materially enough to disqualify a priest from his duties. The Aramaic word *thamir,* according to him, did not signify loss of eyelashes, and the meaning of the word was not clear to him.

That weeping was considered one of the main causes for loss of the eyelashes is testified by the patriarch Gamliel, who claimed that he lost his eyelashes overnight because he wept over the death of a neighbor.[104] Rashi (who died in 1105), the well known Talmudic commentator, was not satisfied that one could lose the eyelashes overnight, and he read in the text that the moisture of the lids from constant weeping turned the eyelashes into the conjunctival sac, producing what is known as trichiasis.

Conjunctivitis Simplex.—Owing to the lack of knowledge of ocular pathologic conditions and to a belief in the humoral theory (i. e., that all diseases of the eye are caused by certain abnormal humors descending from the brain to the eye), the ancient physician could see no distinction between the various forms of conjunctivitis; they were different in both etiology and symptomatology. Antiquity included under ophthalmia all the forms of congestive ocular diseases. R. Judah, when asked for which disease of the eye one may violate the Sabbath Day (without stating the name of the particular malady),[105] gave five cardinal signs: mucous discharge, inflammation, pain, epiphora and hemorrhage.[106] Another Talmudic Rophe mentions concretions between the eyelids which glue the lids together,[107] causing disturbance of the function of the organ. The Talmudic description of catarrhal conjunctivitis recalls to mind the history of the disease as given by Hippocrates. "If the humors that enter the eye," said the father of medicine, "are of a strong and acrimonious character, they will inflame the eye, ulcerate the lid and even excoriate the cheek and other parts with which they come in contact. They cause heat, pain,[108] external burning until the humors are concocted and made thicker, and concretion are formed around the eyes. In severe cases of ophthalmia sometimes rupture of the tunics will occur, especially when exposed to cold wind. Ophthalmia in a warm climate is of a mild character." [109]

104. Sanhedrin 104B.

105. No name for ophthalmia is given in the Gemara. The neo-Hebraic form is Daleketh-halahmith.

106. Abodah Zara 28b. Bezah 22a.

107. Nidah 67a. Mikbaoth 9, 2; -4.

108. In this description of ophthalmia are given the four cardinal symptoms of Hippocrates for inflammation, i. e.: rubor, redness of tissues; tumor, swelling: calor, heat, and dolor, pain, to which is added a fifth sign by Galen, disordered function. It should be noted that the Talmud mentions all of these symptoms.

109. Hippocrates: The Air, Kuhn, Leipzig edition, pt. 1, p. 527.

The following pathologic conditions of the eye may be present in cases of ophthalmia or may afflict the ocular organs independently of any other diseases:

Hyperemia (Kadcha) of the Conjunctiva.—*Kadcha* appears to have been a common condition of the eye characterized by inflammation of the conjunctiva with little or no discharge.[106]

Hemorrhage (Dama).—Hemorrhage of the conjunctival sac is mentioned in the Talmud, but appears to have been very rare as very little is said about it.[106]

Subconjunctival Hemorrhage (Simuka).—*Simuka* is described as bright red patches of greater or lesser size involving the bulbar conjunctiva. Etymologically, *simuka* means red.[106]

Trachoma or Ophthalmia Granulosa.—This condition received the name "Egyptian ophthalmia" at the end of the eighteenth century (1793) when the soldiers of Napoleon in Egypt, most of whom were suffering from trachoma, returned to their homes to spread the most virulent epidemic of trachoma in Europe.[110]

Egypt of antiquity possessed a healthy and salubrious climate, as testified by Tacitus' "Corpora hominum Salubria et ferentia laborum" [111] and earlier writers such as Hippocrates and Herodotus, who praised the wonderful climate, the cleanliness of the inhabitants of Egypt and the idealness of the place as a health resort.[112] For some reason, however, there existed considerable disease of the visual organs. Even before the Hebrew exodus (3400 B. C.), the oldest medical document, known as the Papyrus Eber, (1500 B. C.) describes a form of ophthalmia differing from the ordinary form by its profuse purulent discharge and other symptoms under the term "dripping eyes." [112] The Greeks as well as the Romans left no positive description of trachoma, so that the very name of the affliction is in doubt. One can hardly agree, however, with the eminent ophthalmologist and historian, J. Hirschberg, in his statement, "Dem klassischen Alterthum war der Begriff der ägyptischen Ophthalmologie ganz fremd" (To classic antiquity the conception of Egyptian ophthalmology was totally foreign). Indeed, there is much evidence that trachoma was well known to the physicians of Greece, Rome and Palestine long before the present era.[112]

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110. Scientific investigation revealed that the epidemic of trachoma transmitted to Europe by the soldiers of Napoleon, which caused a large proportion of blindness, was mixed with gonorrheal infection and not the ordinary type of granular lids that were prevalent in Europe before.

111. Historiae 5, 6. See also: The Annals, Oxford translation, 1892, pp. 2 and 59. Genesis 13:10. Psalms 105:37.

112. Cited by Kotelmann, L. W.: Die Ophthalmologie bei den alten Hebräern, Leipzig, L. Voss, 1910, p. 156.

Hippocrates, in the "Third Book of National Diseases," points out many afflictions brought about by weather conditions. One of these is a chronic congestion of the eyes that is accompanied by pain and growths (granulations), "fig warts," which often destroy vision. About five hundred years later Celsus, the celebrated Roman medical writer, stated, "Among some people the eyes are never dry; a muco-purulent discharge escapes always from the visual organs, and with it is associated a roughness of the lids. Such an affliction makes the life of the individual miserable and in some instances the vision is destroyed." [113] According to Kotelmann, trachoma was known among the Roman physicians as *Aspritudo.* The Roman oculists who had their collyria engraved on stones, which they used as stamps to facilitate writing prescriptions, often had the words *Ad Aspritudo* on the seal.[114]

That Egyptian ophthalmia was transmitted to Palestine at a very early date cannot be doubted. The numerous cases of blindness recorded in the Old and the New Testament were probably due to Egyptian ophthalmia (in Biblical days, as it is in modern times). The people of Palestine, which was on the caravan route to Syria and Babylonia, the great centers of ancient commerce and civilization, contracted the disease through contact with Egyptian travelers. The inhabitants of the plains of the Jordan and the valley of Maggido, through which the great highway to Mesopotamia passed, were the first to suffer.[115]

Those afflicted with ophthalmia granulosa were easily recognized by the people at large, and, according to the Talmud, they were known as *baalei reathan,* men of sight.[116] The technical name, however, was probably *Aineidolphos,* dripping eyes.[117] The symptoms as described in

113. Cited by Kotelmann, L. W.: Die Ophthalmologie bei den alten Hebräern, Leipzig, L. Voss, 1910, p. 156. Hirschberg, J.: Die Ophthalmologie bei den alten Griechen, p. 70.

114. About 150 such seals were discovered in France, Belgium, Germany and England, where military camps of the Roman legions were located. Each legion evidently had its own specialist. The name of the oculist is engraved on many of these seals. The most frequent diseases of the eye against which therapeutic measures were taken were: lippitud, discharge; ciligines, opacities of the cornea, and sufuso, cataract.

115. Granular lids are known to be more prevalent in the low plains than in the mountainous districts. According to Hippocrates, in warm climates diseases of the eye are of a mild type and of short duration. (Cited by Hirschberg.[63])

116. Kethuboth 77b. The euphemism of "bad sight." Such euphemistic expressions are common in Biblical and Talmudic literature. The blind were called "open-eyed" in the Pentateuch, and in the Talmud, "much-lighted" (Genesis Rabba 30).

117. Feigenbaum is convinced that trachoma in the Talmudic days was known by that name (Haayin, 1927, Teil-Abib, p. 63). Some writers, however, believe that Ziran (Bekhoroth 43b) was the Aramaic name for trachoma (Jastrow, Marcus: Dictionary of the Targumim and Talmudim, p. 1281). It is interesting to note that in the Papyrus Eber trachoma is also known as dripping eyes.

the Talmud are profuse discharges from the eyes and nose,[118] the presence of flies on the eyelids,[119] and the escape of saliva from the mouth.[120]

The predisposing causes, according to the Gemara, are uncleanliness, infrequent bathing,[121] unkept hair of the head,[122] living in sandy districts where sandstorms are frequent,[92] improper food,[123] sleeping in the damp open air [124] and bathing in the Dead Sea.[125]

The exciting causes of granular lids are direct contact with the secretion by the hands [125] or by towels,[126] and the most frequent cause, contact with the house fly.[127]

In the Middle Ages the scourge of trachoma in Egypt and presumably in other countries bordering on the Mediterranean Sea was widely spread, and it was impossible to check its progress. The Arabic physicians of the Middle Ages understood the pathologic changes in trachoma and recognized the granulation of the conjunctiva as its chief symptom. Sadili, of

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118. Perlman, M.: Mazia's Notes to Midrosh Horephuoth, Palestine, 1929, Teil-Abib, p. 14. Abul-Casam gives as the predisposing cause of trachoma, abnormal condition of the blood (dyscrasia) and chronic disease of the conjunctiva. Dr. Feigenbaum, who had a large experience with cases of trachoma in Palestine and Egypt, found in many cases a profuse discharge from the nose spreading through the lacrimal canal. In such cases, he observed that the treatment of the disease is more difficult.

119. The marshy districts of Egypt and Palestine are to this day infested with flies; particularly is this true of the banks of the Nile. Thus, said Isaiah (7: 18), "And it shall come to pass in that day, that the Lord shall hiss for the fly that is in the uppermost part of the river of Egypt and for the bee that is in the land of Assyria." I have often watched on the streets of Cairo and Jerusalem the distressing spectacle of flies literally covering the lids of the poor victims with trachoma and feeding on their secretions. Most pitiful was it to see poor babies, whose little arms were not developed enough to rid themselves of these torturing insects. (Gordon, B. L.: New Judea, Philadelphia, Julius H. Greenstone, 1919, p. 90.)

120. Dr. Mazia of Jerusalem, who treated many patients with severe cases of granular lids, noticed its connection with "scrofulous diathesis." In children it is accompanied by enlarged and infected tonsils, by glandular involvement and by the escape of saliva from the mouth.

121. R. Yohanan asked, "Why are there no cases of Baalei-reathan in Babylonia?" He gave as the chief reason, "because they bathe in the Euphrates" (Kethuboth 77b).

122. Nedarim 81a.

123. Pesachim 42a. Erubin 29b.

124. Observers have long noticed diseases of the conjunctiva prevailing among persons who sleep in damp air.

125. Sabbath 108b.

126. Sabbath 147b.

127. R. Yohanan warned "To be careful for flies of the 'men of sight'" (Kethuboth 77b).

Cairo, a prominent physician and medical writer of the latter part of the fourteenth century, complained that the people of Egypt suffered more from ophthalmia than those of any other country, and that it was impossible to get rid of the granulation.[128] R. Meshulom ben Menahem, a Jewish traveler through Egypt (1841), reported a great epidemic of ocular diseases in Egypt. Seven years later, R. Obadiah, of Bartonuro, in a letter to his father, noted the same condition, adding that most of the people of Egypt were stricken with blindness.[129] Such deplorable conditions existed in Egypt and to a less degree in Palestine until recent years.

As late as 1890, Ticho found in Jerusalem the percentage of trachoma 30 per cent among the Jewish and Christian populations and as high as 80 per cent among the Arab population.[130] Mohammed Pasha, in discussing trachoma before the first Egyptian Medical Congress in December, 1902, reported that the percentage of trachoma had lessened in Egypt in the last twenty years from 85 to 57 per cent.[131]

The therapeutic measures taken against trachoma by ancient Hebrew physicians are of an empiric nature (most of the remedies cannot be identified). The bark of nut and fig trees is mentioned,[132] and shavings of hides [132] appear to have been a favorite local remedy. Evidently these therapeutic agents were intended for the purpose of rubbing the conjunctival surfaces. The old Egyptian treatment, according to the Papyrus Eber, was to apply verdigris with onion on the lids.[133] Hippocrates advised rubbing the inner surface of the lids with Milesian wool wrapped around an applicator, until the blood ceased flowing and a thin fluid took its place. Another remedy was cauterization followed by a collyrium of oxide of copper, a plan of treatment that cannot be much improved on even in modern times.

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128. Codex Arabicus 834. Cited by Hirschberg, J.: Centralbl. f. prakt. Augenh. **29**:63 (Feb.) 1905.

129. Cited by Feigenbaum: Haayin, 1927, p. 63.

130. Ztschr. f. Augenh. **32**:368, 1914.

131. Of 11,660 patients with trachoma, 17 per cent had trichiasis, 10 per cent had keratitis, 7 per cent were troubled with glaucoma, and 6 per cent had panus. In 1909, I found, among the school children of the Judean colonies, that 22 per cent were troubled with trachoma in different stages of the disease. I was much impressed to find trained nurses visiting the schools every morning to irrigate the eyes of all pupils, and to attend to those who were already suffering from the disease. The neo-Hebraic form for trachoma is Gareneth, from the Hebrew word Garan, meaning a grain.

132. Kethuboth 77b.

133. Wood, C. A.: (*a*) American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913-1917; (*b*) p. 8577; (*c*) letter hist.; (*d*) vol. 3.

Gonorrheal Ophthalmia (Zablegon[134]).—Ancient medicine did not recognize this form of conjunctivitis as a special malady of the eye, although gonorrhea of the genital organs of both sexes was well known and described in the Book of Leviticus,[135] and hygienic precautions were suggested to prevent the spread of the disease.[136] Nothing, however, is said about gonorrhea of the eyes. In Talmudic days, however, that dangerous condition of the eye appears to have been recognized as a special malady. A priest who was troubled with a blennorrheal discharge was not eligible to perform his priestly duties.[137] Evidently, he was considered to be troubled with the same disease as blennorrhea of the urethra. The neo-Hebraic form for gonorrheal ophthalmia is *Neked-haziba.*

Ophthalmia Neonatorum.—No description of acute purulent conjunctivitis of the new-born is found in the early Hebrew and Greek writings. Andrea concluded that that form of conjunctivitis did not exist in ancient times,[138a] but early medical records in India reveal that it was known as far back as the fifteenth century B. C. The Ayun-Veda, the book credited to Charak and Sustra, mentioned this pathologic condition and described its etiology as due to faulty mother's milk.[139a] That gonorrhea of the new-born was known to Hebrew physicians of the Gemara seems probable, for one finds that expectant mothers were warned to abstain from certain food that would cause dripping of the eyes. One vegetable known as *tachle,* translated water cress, and grown among flax was said to cause dripping of the eyes in the new-born.[140] No special treatmtent for this form of ophthalmia is given. The neo-Hebraic form is *Zibath-hayeludim.*

Zugdus.—This is a condition of inequality of both eyes, or certain parts of both eyes, such as aniso-oculi, anisocoria, or anisopalpebra.[141]

Ankyloblepharon (Damzer)[98].—This is a condition in which the margins of the two lids are adherent, and when complete it results in loss of vision. Antiquity classed it under the term of blindness. The

134. Zablegon is derived from the Hebrew word Zab (to flow—to drip). The discharge is described as a white mucus, resembling barley water (Nidah 35b).

135. Leviticus 15:1; -24.

136. Persons who came in contact with the patient, his bed, furniture, clothing and the place which he occupied were declared unclean[135] and had to undergo ablution and have his things thoroughly washed. The patient himself had to wait seven days before he was declared clean.[135]

137. Megilah 24b.

138. Andrea: (*a*) Die Augenheilkunde des Hippocrates, Madgeburg, Haenel, 1843, p. 80; (*b*) p. 61.

139. Kotelmann, L. W.: (*a*) Die Ophthalmologie bei den alten Hebräern, Leipzig, L. Voss, 1910, p. 220: (*b*) p. 366; (*c*) p. 362.

140. Kethuboth 60b.

141. Bekhoroth 43b.

treatment for *Damzer,* in the Talmud, is to pass a probe (preferably silver) between the lids. This treatment is also recommended by Celsus and is thoroughly modern.[133b]

Nystagmus (*Anai-Neida*).—This is defined as a short, rapid, involuntary movement of the eyeball, usually affecting both eyes. Rashi terms it *Thruthos.*[98]

Strabismus (*Soche-Shemesh*).—Etymologically speaking, *Soche-Shemesh* means "gazers at the sun," but the term is used as a euphemism for strabismus. The ancients did not recognize latent squint, and used it synonymously with diplopia.[142] The Tosefta [143] explains *Soche-Shemesh* as a disease in which one sees objects in the room and on the ceiling at one glance, a condition of vertical diplopia.[144] *Limiz* is another term for strabismus and probably refers to horizontal diplopia.[145] According to Paul of Aegen, an ancient authority on medicine, it is due to cramps of the muscles of the eye. "Strabismus est convulsoria effectio musculorum bulbum moventium." (Strabismus is a convulsive effect of the muscles that control the bulbar movements of the eye.[146]) Galen gives the cause of squint as paralysis of the muscles of the eye. "Strabismus est paralysis musculorum oculi." Aristotle, and later Celsus, believed that squint is a sequel of epilepsy of childhood, because in epilepsy the eye turns in and out. The Talmud ignores entirely the etiology of squint. The neo-Hebraic form *pzilah* is borrowed from the Aramaic word *pozal* (to turn, to twist).[147]

Pterygium (*Nachas*).—This pathologic condition of the eye was known to the ancient physicians. Celsus [148a] describes it and Galen mentions it. It was known to the writers of the Talmud as *Nachas,* because of its tendency to creep on the cornea.[149]

Phthyriasis Palpebrarum (*Kinemeth*).—This is still a common discase of children in Egypt and Palestine. The margins of the lids and the eyelashes become covered with black nits (pediculus pubis). The condition is marked by redness and itching of the borders of the lids. Pedicula were often known as *Kinei beth-hashehi* (lice of the armpit).

142. Zivor, Bekhoroth 44a.

143. A code of laws compiled after the Mishna.

144. Tosefta Bekhoroth 5:3; 73b.

145. Dechoze-Lemalah (hypertropia) and Dechoze-Lematah (infratropia). Bekhoroth 44a.

146. Hypomnema, English translation by Adams, London, Syndenham Society, 1844 and 1848, lib. 3, chap. 22.

147. Psikta Rabba 14. In the Papyrus Eber it is also known as (Nhet M. Mrte) turning of the eye.

148. Celsus: (*a*) De arte medica, lib. 7, chap. 7; (*b*) chaps. 6 and 8.

149. Bekhroth 6:21; 4:2; 38a.

Celsus was the first to use the term *phthyriasis,* and he attributed it to bad habits of the body. He said: "It causes flux and gum (scales) and the eyes often become ulcerated. It occasionally destroys vision." The treatment, according to Celsus, consists in purges by crysters, clipping the hair of the head,[122] exercise and strong gargles.

Foreign bodies in the eye are frequently referred to in the Old and the New Testament as well as in Talmudic literature.[150]

DISEASES OF THE EYEBALL

Macula (Dak) on the Cornea.—Dak has been described as an opacity of the cornea appearing as a gray spot on the pupil and interfering with vision. The Talmud says that if this condition clears up within thirty days, the disease should be considered cured and not likely to reappear. Otherwise, the priest troubled with a *Dak* on the cornea is ineligible for priestly duties. "One must not confuse," warns the Talmud, "mucus on the cornea that can be removed, and a macula that is adherent."[151] Mimonides holds that an umbilicated corneal cicatrix comes under the same category as a macula. Two kinds of *Dak* are described: one that is loosely connected to the cornea and is movable, and the other that is tightly adherent to the cornea.[151] The first might be a panus.

Leukoma (Chvarvar [151]).—This is described as a white spot on the cornea. It differs from *Dak* in the denseness of the opacity. The entire pupil or part of the pupil appears white, and for that reason was sometimes described by Ancient writers as a white star. No etiology is given in the Talmud for this corneal disease. Rashi's description of *Chvarvar* is that there are "small pinpoint opacities in the cornea" (probably keratitis punctata), but nothing is said as to where the white spots are located. This disease, according to the Gemara, is chronic if it lasts more than eighty days.[152]

Interstitial Keratitis (Barkith).[151]—This term is probably derived from the Aramaic word *Barkai,* which signifies a morning star.[153] Because of its grayish appearance it is sometimes mistaken for a cataract,[154] which is often known as a "gray star." The treatment administered for cataract was often prescribed for *Barkith.*

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150. "They shall be pricks in thy eyes" (Numbers 35:55). "Thorns in thy eyes" (Joshua 23:13). "Let me pull out the moat from thine eye" (Airechin 16b; Baba Bathra 15b). "Take out the splinter from thine eye" (Airechin 16b; Baba Bathra 15b).

151. Leviticus 21:20. Bekhoroth 38a; 38b; 44a.

152. Footnote 151. Curiously enough, in the Papyrus Eber leukoma is also known as white (sht.).

153. It may be derived, however, from the Hebrew word Barak (lightning) (Deuteronomy 32:41. Exodus 19:16).

154. Sabbath 78a. Tosefta Sabbath 6:7. Giltin 69a.

Hypopion; Keratitis.—Pus within the anterior chamber was known under the name *liphluph,* and is referred to in the Mishna.[155]

Coloboma (*Thebalul*).[156]—Etymologically speaking, *Thebalul* is a mixture of colors of the iris and sclera and is understood to be two congenital conditions: a coloboma of the iris and sclera, and also a condition in which the sclera covers part or the entire pupil.[157] The first is not considered a defect by the Mishna as long as the pupil is clear, even though it cuts through the iris and part of the sclera; the second, because it obstructs the pupil, is a defect,[158] and if a priest possesses such a malady he is incapacitated for priestly duties.[159] Modern medical books in Hebrew use *Thebalul* for panus because of its phonetic similarity to the Arabic *Sebel* (panus),[160a] but the text of Bekhoroth 38a leaves no doubt that it is referring to coloboma of the iris and sclera and also to coloboma of the cornea covered with white sclera.[15d]

Cataract (*Mayim hakebuim*).[161]—*Mayim,* the Hebrew word for water, is applied to opacities of the lens or its capsule. The ancients and people of the middle ages, up to the beginning of the eighteenth century, believed that the so-called gray star was a result of injurious humors of the brain flowing through the hollowed optic nerve into the eyeball, settling between the iris and the lens. In the earliest records of ophthalmology, known as the Papyrus Eber, cataract is called the mounting of water in the eye (*Kt. nt dr. acht nt mu m mrte*). Other writers, such as Hippocrates and Galen, called it by a similar name. The Arabs called it *Al-ma an-masil fel-ain,* the flow of water of the eye, and the Romans knew it by *affuso oculorum.* "If one strikes a slave upon the head," says the Talmud, "and the water of his head flows down the eye and blinds him that slave must be liberated."[162] The blindness mentioned is evidently cataract. Ancient ophthalmologists, owing to their improper knowledge of the pathology of cataract, thought that cataract was caused by a fluid running down from the head and lodging itself between the iris and the lens. Accordingly, a flow of tears was a good omen in the case of cataract, because by releasing the fluid from the eyes it would do away with the cause of the disease. "Thus,"

155. Mikbaoth 9:2, 4. Nidah 67a. This condition was recognized in the Papyrus Eber under the name of Seq t' ft M. Mrte, literally meaning "closing of the pupil."

156. Bekhoroth 38b.

157. Bekhoroth 44a; 38b.

158. Bekhoroth 38a; 38b.

159. Sifra 95c.

160. Feigenbaum: (*a*) Haayin, 1927, p. 52; (*b*) p. 93.

161. Bekhoroth 38b. (Constantine Africanus is said to have suggested the term cataract. His work "De Oculis" is a large compilation of Arabian ophthalmology.)

162. Baba Kamma 98a.

said Rabbi Simon ben Elizer, "when the eyes tear, the prognosis is good, and when the eyes are dry, the chances for improvement are bad." [158] Another cause of cataract is traumatism.[163] The treatment for cataract was to abstain from drinking water [164] and eating food that would make one thirsty, such as fish, in order to keep away the fluid of the body. The neo-Hebraic term is *Yarud* (to descend). In the Talmud, however, *yarud* apparently stands for exophthalmos.[165]

Glaucoma Ophthalmia; Arthritica; Sea Green Star (Mayin Yerukim).—This common disease of the eye, which was responsible for much blindness in the olden days, was not recognized by ancient physicians. Even the congestive type that can be perceived by inspection and touch was recognized only by a few ancient writers. The name glaucoma was generally applied to a greenish discoloration of the pupil, sometimes noticed in the later stages of the disease and popularly known as green star. The condition was often mistaken for cataract or for inflammatory diseases of the eye. Hippocrates mentioned it only once, under the name *glaucosis,* and then in connection with cataract. In old Greek and Roman literature glaucoma was applied to a pathologic condition the chief symptom of which was a light blue pupil.

A pseudo-Galenic writer defined glaucoma as a condition in which the natural fluids of the eyes are changed into a clear blue color and are accompanied by complete blindness. The distinction between blindness and glaucoma was the color of the pupil. In blindness it was dark, and in glaucoma it was blue. Both conditions were incurable, but when the eye retained light perception it was called *hypochyma.* Simple glaucoma was not recognized at all. The early records on the subject leave the impression that the chief symptom of glaucoma, which is interocular tension, was not recognized.[166]

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163. Tosefta Bekhoroth 4:4.

164. The one who drinks water from a large receptacle promotes the growth of gray star (Pesachim 112a). According to Hippocrates, cataract is an incurable disease the visible symptoms of which are: light blue, dull blue or silver gray pupils. When such colors are present nothing can be done for it (Hirschberg [footnote 63 p. 86]).

165. Rashi Abodah Zarah 28b.

166. It is evident that the Hippocratic writers laid much stress on the coloring of the pupil in the diagnosis of glaucoma. They recognized, however, that glaucoma is a bilateral condition when they stated that a sea (Mediterranean) colored pupil in one eye is certain to have an injurious effect on the other eye. To Dr. Mackenzie (1830) is due the credit of recognizing this most important symptom, and of suggesting a measure of relief, which was repeated paracentesis of the cornea. Ophthalmologists are particularly indebted to von Graefe, who in 1856 devised iridectomy for the first time for glaucoma. This was the most important discovery in ophthalmology. It must be borne in mind that formerly every patient with glaucoma was forced to sink into the night of blindness and now, thanks to skilful surgical intervention, this condition is curable in the majority of patients.

Staphyloma of the Cornea (Enab).—This disease is so called because of its resemblance to a grape. It is the bulging of a part of or of the entire cornea caused by a corneal ulcer or by interocular distention. It seems to have been a frequent ocular condition in ancient times. Galen, who called it *tuberculum,* first described it and named it the grape-shaped tumor. It is usually white, but may be dark or gray. Celsus stated, "In the eye itself, the external coat is sometimes raised through either rupture or relaxation of some of the internal membranes and resembles a raisin stone in its form." [167] The Talmud adopted the same name and called it *Enab,* from the Greek *staphyloma.* This name appears also to be popular among the Arab physicians, who termed it *Inaba.* It is sometimes referred to in German books on ophthalmology as *Trauben-Auge.* Rabbi Safro called it by the Aramaic name *Anoibatha* and termed it the messenger of death.[168] (The Rabbi was probably thinking of Aristotle, who said that diseases of the eye were dangerous to life because all the nerves and vessels of the eye are connected to the heart; the heart was taken as the seat of the soul.[133c]) He suggested the application of a herb called *Tignah* soaked in honey. Another remedy suggested is a split grape of the same color and size placed over the affected eye. Celsus (25-50 A. D.), after discussing operative intervention, advised the application of the white of an egg.

Ophthalmomyosis; Parasitic Ophthalmia.—This disease is a rare pathologic condition caused by animal parasites entering the eye through the nose or lacrimal canal and thence going to the brain, causing damage to the visual centers of the brain, and is mentioned in the Talmud.[132] The treatment as outlined is removal of the parasite from the brain.[169]

Exophthalmos (Merida).—This is described as bulging of one or both eyes.[170] The elder Pliny [171] mentioned a condition under the name *Prucidentia Ocularum.* Hippocrates alluded to it, but only as a grave symptom of certain febrile diseases.[138b] Celsus also referred to it as a bad sign if it manifests itself in certain afflictions of the body.[148b] The occurrence of exophthalmos appears to have been attributed by the ancients to an increase of fat in the orbital cavity.[172] Reference is made in the Book of Psalms to exophthalmos goiter.[173] The seriousness of this disease in the opinion of Talmudic authorities may be gathered from

167. Introduction 16, 14, 774, Kuhn, Leipzig edition.

168. Abodah Zara 28b.

169. Mazia's Notes to Midrash Horephouth, Teil-Abib, 1929, vol. 2.

170. Rashi, famous commentator on the Talmud, renders Merida, a rebel.

171. Natural History 34, 50, cited by Preuss.[15e]

172. "Their eyes stand out with fatness." Psalms 73: 3.

173. "But the Lord will give you a trembling heart and a failing eye and a sorrowful soul" (Deuteronomy 28: 65).

Tractate Sabbath, which states that a rebellious eye was permitted to be cured on the Sabbath Day.[174]

The definition, however, that *Merida* is identical with exophthalmos is not satisfying. A story is told of a servant in the house of Mar Samuel, who took sick one Sabbath Day with violent pain and inflammation in one eye. The employers thought that her condition was trifling and that the domestic was hypersensitive; as it was the Sabbath Day, they did not think the case serious enough to bother about it. The condition became aggravated during the day, and toward evening the eye became perforated.[168] Of course, the Rabbis ordained that from then on all patients with such trifling cases should be treated on the Sabbath. From the description, it does not appear to have been a case of exophthalmos.

Phthisis Bulbi (*Ukla*).—Shrinking of the eyeball was ascribed to chronic ophthalmia and was noticed by ancient physicians. Celsus describes it and gives as its cause *Lippitud* (ophthalmia, particularly in its later stages, when an acrid fluid or gum covers the eye). Other causes for this disease given by Celsus are weeping, badly cured wounds of the eye, anxiety and a general run-down condition.[175] The treatment suggested by Celsus was nourishing food, particularly milk, collyria and removal of the causes, if known. Many cases of phthisis bulbi are observed in Palestine and Egypt to this day.

AMAUROSIS

The Hebrew term for blind is *Iveir;* the Aramaic form is *Suma.* These terms embrace all cases of blindness, whether due to a faulty state of the media, or a disease condition back of the eye in the visual centers, the optic nerve or the blood vessels or whether the condition is secondary to many constitutional conditions. The Talmud often uses the euphemistic expression *Shagi-nahar* (brilliant vision). The term *Iveir* is often used in the figurative sense, such as "The bribe blindeth clear-sighted."[176] In antiquity blindness was attributed to a divine punishment for sin.[177] This is well illustrated in the New Testament, "He saw a man blind from birth," and his disciples asked him, saying, "Master, who did sin, this man, or his parents, that he was born

174. Jerusalem Sabbath 14; 14d. Abodah Zara 28b; 40.

175. The Book of Zacharia (1412) refers to consumption of the eyeball. "And their eyes shall consume in their holes." The Hebrew tamach is rendered by LXX and Vulgate; "to consume, a condition where the black of the eye disappears." Unkelos and Peshito translate it Dub (to dissolve or melt). Kahut Aruch Completum, letter Ukla Beza 22a.

176. Exodus 23:8. In Arabic, a distinction is made between monocular blindness and binocular blindness. The first was termed auur, and the second, aema.

177. Deuteronomy 28:28. Zephaniah 1-17.

blind?" [178] Nahum considered his loss of vision as a divine punishment because at one time he was not prompt enough to give food to a beggar.[179]

That the loss of vision was a common defect in Biblical days is not doubted. Both the Old and the New Testament abound with cases of amaurosis; indeed, they were so numerous that special laws were necessary for the protection of the patients.[180] The ancients regarded it as the lowest form of degradation. The blind, crippled and lepers were considered outcasts of society; they were entirely excluded from social life and were kept quarantined outside the city gates. As a result, they became paupers [181] and a menace to travelers and passers-by. Hence, one reads that when David besieged the Jebushites of Jerusalem, the blind and the crippled were so numerous that he was compelled to take stringent measures against them.[182] Even at a later date, during the first few centuries of the present era, people looked on the maimed, especially the blind, as being debased in character. The old Greek belief that bodily defects are an expression of low spiritual and moral status was responsible for similar diction in the Talmud.[183]

Thus one is told that the patriarch Rabbi Judah, when he and Rabbi Hyiah came to a certain town and inquired whether there were any learned men in the community whom they could visit, was directed to a blind scholar. The patriarch was ready to visit this scholar, but his colleague said to him, "Do not disgrace thy excellence." The patriarch, however, did not heed the warning of his friend and he entered. When he was ready to leave, the blind man, very much honored by the visit, remarked, "You have honored, by your audience, one who is seen but sees not; ye shall be blessed and acceptable by one who sees but is not seen." [184] Because the blind were looked on as outcasts of society, the Rabbis took stringent measures to counteract the general feeling of the people toward them.[185]

In view of the numerous "cures" of the blind recorded in sacred literature, Biblical critics are of the opinion that the word blind was used rather loosely in the Bible. Closure of the lids resulting from

178. John 9:1; -2. ("Bar Jesus became blind because of his wickedness."—Acts 13:2.)

179. Nahum ish-gam-zu. Taanith 21a.

180. Deuteronomy 27:18.

181. Matthew 9:27; 12:22; 20:30; 24:24. John 5:3.

182. II Samuel 5:6.

183. Sanhedrin 105a. Taanith 24a.

184. Hagiga 5b.

185. Jerusalem Peah, end of chap. 8.

inflammation or ptosis and diseases of the cornea temporarily interfering with vision were termed *Iveir,*[186] although such conditions frequently disappeared in the natural way.[187] That hyperbolic terms were used by Biblical writers cannot be doubted, and it is likely that antiquity has applied the term loss of vision for impairment of sight. Indeed, even as keen an observer as Hippocrates used such terms in cases of temporary diseases of the eye, interfering with sight, when he said that "Blindness will disappear providing no blemish has been afflicted on the cornea." Be that as it may, there can be no question that ancient physicians have applied the term amaurosis not as an end-result, but as the disease itself. While there can be no question that such diseases as retrobulbar neuritis, albuminuric neuroretinitis, optic neuritis and other forms of organic diseases existed in ancient times, diagnoses could not be made. Therefore, the name blind was applied to all pathologic conditions visible or invisible to the naked eye.

Temporary Amaurosis (Shabriri).—This is a condition of the eye in which one is subjected to sudden temporary blindness. Two kinds of *Shabriri* are described: day *Shabriri,* hemeralopia, and night *Shabriri,* nyctalopia. The cause of day *Shabriri* is a sudden strong light flashing into the eye, as the bright sun shining through a crack in a dark room or through a dark cloth.[188] The cause of night *Shabriri* is given as the drinking of water from creeks, brooks, lakes and, particularly, stagnant pools. The cause of this blindness was probably due to a severe form of malaria.[160b] Pernicious malarial fever prevailed in many sections of Palestine, and the parasites in the red corpuscles caused hemorrhage in the retina and blindness by breaking down the quality of the blood. Thus, the Rabbis who prohibited the drinking of water from infected pools took a far advanced measure to check the prevailing scourge.[189] The popular belief, however, was that a certain demon by the name *Shabriri,* who dwelled in the water, was responsible for the blindness.

The question of whether or not the loss of sight has a compensatory effect on other sensory organs such as hearing, touch, taste and smell (a subject much discussed by ancient as well as by modern observers) was decided in the affirmative by Talmudic authorities. In the blind

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186. Some Old Testament students believe that the mere covering on the cornea was stigmatized Iveir and referred to the Hebrew etymology of that term. If not punctuated, it reads "or," meaning a skin in front of the eye (Paul of Aegan makes a differential diagnosis between amaurosis and glaucoma. In the first, the color of the pupil is not changed. In the second, the color of the pupil is changed.)

187. This opinion is voiced in order to explain certain miracles mentioned in sacred literature.

188. Gittin 69a. Yoma 28b.

189. Pesachim 112.

all the special senses are more developed than in the seeing persons.[190] "A blind child can recognize the mother by the odor of her breasts and the taste of the nipples." [191] A story is told about the blind Rabbi Sheshest, who on one occasion, while the guest of the Exile-Arch, distinguished the footsteps of the Persian King from the steps of his guards by his dignified walk on the sands.[192] On another occasion, while dining at the Exile-Arch, he did not partake of a certain food. When he was asked the reason, he replied that he could smell the leper in the food.[193]

Another interesting observation made by the Gemara is that the blind have excessive appetites: "They eat, but they do not fill up." The "filling up" is influenced by the visual organs.[194] This Talmudic statement is corroborated by the observations of superintendents of institutions for the blind, who also noticed this strange phenomenon in the blind.

OPTICS AND REFRACTION

It is problematic whether or not optics was known in the first four centuries A. D., particularly in its relation to refraction of the eye. The Talmudic reference to *Ispeclaria-hameira,* translated a magnifying glass,[195] might just as well be applied to a reflecting object, instead of refracting media. The same doubt may be cast on the statement of Pliny, who records that Nero viewed the games of the gladiators through a glass (*Sumaragadus*); he probably was referring to a large concave mirror from which the scenes were reflected. There are no other records, in Greek or Roman literature, which mention that spectacles

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190. de la Sizeranne, Maurice: Blind as Seen Through the Blind Eyes, translated from the French by Park Lewis, in American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913-1917, p. 1209. In modern verse:

> This fellow must have a rare understanding,
> For nature recompenseth the defects
> Of one part with redundance in the other
> Blind men have excellent memories and the tongue
> Thus indisposed there is treasure in the intellect.
>
> —Shirley.

191. Kethuboth 60a.

192. Berachoth 58a. Bezah 14a. In this connection, it will be interesting to read a book by a modern blind author who gives a graphic description of objects and things as he perceived them. Levy, W. Hanks: Blindness and the Blind, London, 1872.

193. Gitten 67b.

194. Yoma 74b.

195. "All prophets," says the Talmud, "looked through spectacles that did not increase the light; but Moses looked through spectacles that did increase the light." Yebomoth 49b. Rosenfeld, Adolph: Das Auge in Bibel und Talmud, Berlin, 1892, p. 25.

were in use during the early years of the present era in Europe or the Near East. It is known, however, that a Chinese emperor (2283 B. C.) employed glasses to observe the stars. These were framed in tortoise-shell rims, because the tortoise was sacred to the Chinese, and the wearing of such frames was inductive to good fortune and long life. The glasses themselves were ground from transparent rock crystal or perhaps amethyst found in the Holy mountains of the Celestial Empire.

Yet one who examines the almost unintelligible minute inscriptions of the Egyptians, found in the British Museum, must conclude that the Egyptians did use a magnifying glass for their engravings and writings. Remnants of convex glasses were discovered by archeologists in ancient Nineveh and Pompeii, and while most of the finds are not conclusive, as the foci of these glasses are too short to have been used for eyeglasses, yet there are some that could be used by skilful workers in their minute and exquisite work on gold and precious stones.

The use of spectacles was first confined to elderly scholars and theologians whose eyes were strained from reading and copying old manuscripts. The wearers of glasses were looked on as superior persons; hence, the anachronism of a medieval artist who painted a portrait of St. Paul wearing glasses and reading a book. (The spectacles are situated low down on his nose.)[196]

Artificial eyes made of gold are mentioned in Talmudic literature.[197] Kotelmann points out that the use of gold eyes has long been a practice among the Egyptians. Many mummies were found with gold eyes, and the Talmudic record of artificial eyes is an old device.[198] Hirschberg, however, doubts that such was the practice in Talmudic days.[199] Preuss remarks that the gold eyes found in Egyptian mummies were the result of a postmortem operation, and were put in before the masks were placed on the dead. Such a practice was also common among the

196. In Germany, as far back as the fifth and sixth centuries, the aristocrats were wearing frames without glasses, evidently eager to assume a scholarly aspect. This practice survived until late in the nineteenth century. An inferior rim was worn by those in the presence of superiors. In Italy, for some reason or other, the wearing of glasses was not considered a mark of distinction. Salvino Aramato, the reputable inventor of spectacles (1290), was ostracized for his device. The inscription over his grave in the cemetery of Florence reads, "Here lies Salvino Aramato, Inventor of Spectacles. May God Pardon Him His Sins."

197. Jerusalem Nedarim 9:8, p. 62. Nedarim 66b.

198. Kotelmann 139b.

199. Hirschberg (footnote 63, p. 27). He cites Jastrow as saying that an ignorant manuscript transcriber had mixed eyes for teeth, since it is well known that gold crowns on decayed teeth are an old device (Sabbath 64b. Jerusalem Sabbath 6:5. Nedarim 66b).

Greeks and Romans.[200] The Hebrews, however, could not be accused of postmortem surgery, because such operations are strictly prohibited. In absence of positive evidence as to the originator of artificial eyes, the credit for them goes to Ambrose Pare (1517-1590).

The writers of the Gemara knew of senile failure of accommodation[201] and of errors of sight.[202] They speak of long sight and of short sight;[203] they understood the principles of convergence, and the difference between human vision and animal sight;[204] they observed the pupillary reaction to light.[205] R. Gamliel devised a tube; by cutting out the fields, he was enabled to see at a distance.[206] The range of vision for far distance is given as 2,000 cubits.[207]

ANCIENT THERAPEUTICS

In adhering to the theory that the humors under certain conditions are responsible for ocular pathologic changes, one cannot expect to find in ancient therapeutics much valuable information concerning treatment for diseases of the eye.

Therapeutics of antiquity has little of the rational element in the Pharmacopeia. It may be divided into two classes: natural and supernatural. The first embraces drugs, herbs and other therapeutic measures that had as their object the treatment of disease; the second included prayers,[208] divination, incantations, magic, amulets, cameos, charms and talismans. The natural remedies were the practice of the physician; the supernatural, those of the medicine man. At times, however, the physician would take into his dominion the supernatural therapeutic

200. It must be borne in mind that among the Egyptians the preservation of the body was a sine qua non to the resurrection of the individual. Egyptians who died eyeless had the embalmers correct their defect by fitting in artificial eyes, in order that the rebirth should not be defective in any way.

201. Rabbi Nachman said, "Until 40 years, one may better his sight by using a collyrium; after that age, nothing can be done" (Sabbath 151b. Tana-debei Eliyahu Zuta, chap. 3).

202. Sabbath 151b.

203. Jewish Encyclopedia, letter eye.

204. Genesis Rabba 88. "The human is able to focus both eyes, while the animal is not."

205. Zohar, pt. 1, p. 170.

206. Erubin 43b.

207. Footnote 206. Pliny cites the case of a man who could distinguish objects at a distance of 135 miles. Standing on a promontory of Sicily during the Punic Wars, he could see the fleet coming out of Carthage, and could even identify the number of vessels. The statements of R. Gamliel should not be taken as an exaggeration, for in Palestine, owing to the atmospheric conditions, one can see and hear from a very great distance.

208. R. Alexander said, "In the name of R. Hyia bar Abba, the sick will not recover unless his sins are forgiven."

measures to make doubly sure of his cure, and possibly also to prevent the medicine man, his competitor, from getting the better of him. The physician resorted first to natural cures and made the supernatural an auxiliary affair.[209]

In the case of *Shabriri* (night blindness), the prophylaxis suggested was to abstain from drinking polluted water, and the local application prescribed was the ashes of a burnt (seven colored) scorpion, either in powdered form or mixed with a collyrium. The popular belief credited the disease to demons. Therefore, the patient was also treated with anti-demonic measures. He had to repeat a formula that consisted in dividing the word *Shabriri* (the demon causing the disease) into syllables, such as *-SH-SHA-SHAB-SHABR-SHABRI-SHABRIR-SHABRIRI,* followed by the following incantation: "My mother told me to beware of *Shabriri.*" Similar to this was the formula for *ABRACADABRA,* used as a remedy for intermittent fever or for inflammations. The patient was made to wear an amulet around the neck and the word *ABRACADABRA* was written in the form of an inverted cone.[210] The idea was to force the spirit to relinquish its hold on the patient.

In Palestine and Babylonia, in order to remove a boil, one had to repeat the following formula: *"Baz Baziah, Mas-Masiah, Kas-Kasiah, Sparlai, Vamarlai."* [211] The incantation was, "Yea angels that come from Sodom to heal painful boils, let the color become red, let it not further spread, let the seed be absorbed in the belly, as a mule does not propagate itself in the body of M, son of M, so let not evil propagate itself." Such superstition was not confined to Palestine alone. In Rome, when Marcius Parcius Cato, a censor, who also practiced medicine, desired to reduce a dislocation, he recited the following: *Hanat, Banat, Ista, Pista, Sista, Damunata.*[212]

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209. When preparing a salve for a patient, the patient should repeat the following incantation, said Aetius: "The God of Abraham, the God of Isaac, and the God of Jacob, give virtue to this medicament." When a patient had a bone stuck in his throat, the physician should seize the patient by the neck and cry out in a loud voice, "As Jesus drew Lazarus from the grave and as God has drawn Jonah from the whale, thus Blassius, the martyr, and servant of God, command the bone to go up or down."

210. ABRACADABRA
BRACADABR
RACADAB
ACADA
CAD
A

This formula was first mentioned by Serenous Stemonicus, physician to Emperor Caracalla, whose work "De Medicina Praecepta" was admired by Emperor Alexander Severus. It is erroneously said that ABRACADABRA is a cabalistic symbol and is a corruption of the Hebrew HABRAHA DABAR (bless the object) The cabala has nothing to do with it.

211. Sabbath 67a.

212. Cited by Preuss, Julius: Biblisch-talmudische Medizin, Berlin, S. Karger, 1911, p. 165.

Thus it is seen, when ancient medicine called to its aid magic to rout the demons, the methods employed were primitive, if not ridiculous.

Among popular therapeutic agents used in ocular practice by ancient physicians was spittle. It was taken from a healthy person before breakfast.[125] One reads in Mark, "And he took the blind man by the hand and led him out of the town, and when he had spit on his eye and put his hand upon him, he asked him if he saw aught, and he saw things clearly." [213] The cure of inflammatory diseases of the eye with spittle was also popular among the Romans and Egyptians. Tacitus states that "the eyes of Emperor Vespasian were cured with spittle." [214] In Egypt, spittle was used as a solvent for disturbing films in the eye. Spittle is still in use in Eastern countries and in rural districts of Europe for inflammatory diseases of the eye.

In rural England and France, spittle belongs to the midwife house remedies and is responsible for many cases of neglected ophthalmia neonatorum. The spittle of the seventh son of the same wife was particularly effective, and the father of seven sons was in an enviable position, demanding a high price for this much sought remedy. The Talmud prefers the spittle of the first born son.[215]

Next to spittle the ancient writers mention a popular remedy called collyrium. Unlike modern eye lotions, collyrium was made in three different forms (i. e., liquid, semisolid and powder). The Greek collyrium contained as one of its ingredients oxide of antimony. In Palestine collyrias were sold in public market places with other merchandise; the ingredients were not disclosed. Some collyria might have contained dope or poison, for the Talmud warns Jews not to buy it [216] from the heathen for fear it contained poison.[217] It was often made in paste form. Pliny states that a cataplasm collyrium consists of soaked bread, wine, water, milk or white of an egg, in addition to the regular ingredients. It was made up in the form of small bars. In Rome the

213. Mark 8:23. John 9:6, 7.

214. Tacitus 4, 81.

215. Baba Bathra 126b. During my travels in the East, I have seen old women use spittle in cases of catarrhal conjunctivitis. Feigenbaum tells of a practice among the natives of Palestine, who remove a foreign body from the eye by licking the eye with the tongue. Some Arabs would not permit any medication in the eye at all, as they believed that the mucus absorbs the injurious salt from the eye. In Brandenburg, Preuss saw an old woman licking the eyes of a child sick with conjunctivitis. Erlinger found antiseptic properties in spittle. He claims that it contains potassium sulphate (Med. Ztschr., 1895, p. 294 [Berliner Verein für innere Medizin, March 18, 1895]).

216. Nidah b55. "Sama deshanto," a hypnotic lotion, probably an opium derivative, is mentioned in the Talmud (Baba Mezia 83b).

217. Sabbath 109a.

ingredients were stamped on the bars. In Palestine, the bars were sold under the name *Kilirith*.[218] Celsus names ten different collyria and gives the ingredients of each. They seem to have been well known, for in prescribing, physicians identified the different collyria by numbers.[133d]

Dry collyrium (xero-collyrium) was used in powder form, intended to be dusted in the conjunctival sac. The use of the powder appears to have been a painful procedure. Once the famous patriarch, Rabbi, was suffering from some ocular disease and he was advised by his physician to use the powdered collyrium. The Rabbi refused to be treated with this irritant.[219] Mar Samuel, a prominent physician of the third century, had no faith in any of the collyria. He said, "A few drops of cold water in the morning and a general bath in the evening is better than all the collyria in the world."[125] Another popular drug, often spoken of in Talmudic writings, is *Cohol*. According to Kahut, it was a herb that colored the tissue of the eye, somewhat like mild silver protein, and was supposed to strengthen the muscles of the eye.[220] It was applied on the lids and within the conjunctiva. Until 40 years of age, *Cohol* was supposed to have a beneficial effect on the eyesight, but after 40 it had no effect.[221] Another collyrium known as *Puck*, rendered *Stibium*, is recommended for the growth of falling eyelashes.[222]

The gall of reptiles, animals and fish was used for various diseases of the eye, particularly to absorb films on the cornea as in the case of Tobit.[223] In ancient Babylon, the gall of frogs was used in certain diseases of the eye. Diluted reptile gall was used by the Egyptians for absorbing obscuring films.

Blood, particularly that of a bat, was used frequently by ancient oculists. According to the Papyrus Eber, it is a specific in trichiasis. Galen also recommended it in certain ocular diseases. The Talmud finds it efficient in leukoma and proptosis.[224] Bat's blood is a popular remedy in some countries to this day. It is believed that when one paints his

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218. Sabbath 72b; 77b.

219. Babe Mezia 85b.

220. Aicho Rabba 2.

221. Cohol must have been a popular drug. According to Stein, an oculist in Persia and Turkey is called cohol (Turkai 1-66). In Arabia oculists are called Cahalin.

222. Sabbath 109a. In the books of the Prophets, the word Puck stands for a cosmetic women applied to the inner surface of the eyelids in such a way as to produce a narrow black rim, making the eyes appear larger; and consists of a mixture of plumbagin and zinc (II Kings 9:30. Isaiah 54:2).

223. Tobit 6:6. Creighton is of the opinion that the patient with leukoma in the Book of Tobit was not cured by gall, but that the gall was mixed with the burnt liver and heart of a fish and was used for tattooing the leukoma (Kotelmann [139c]).

224. Sabbath 78b.

eyes with this blood he will see at night as well as during the day.[225] Hippocrates' treatment for nyctalopia was the administration of ox liver in honey, purgatives to drive the humors away. Counterirritants, such as strong gargles and sharp masticatories, to drive away the injurious humors or fluxors from the eyes were used. Local applications were seldom employed in acute cases for fear they would increase the fluxors.

Other therapeutic measures for diseases of the eye were phlebotomy, cupping and cauterization. The Hippocratic procedure of cauterization was to put a hot iron over the course of the blood vessels to forestall further fluxation by destroying or obstructing the blood vessels. The Talmudic writers recommended heat for inflamed parts,[226] an old theory being that disease might be cured by remedies which if given to healthy persons would produce diseases;[227] this antedated Hahnemann's theory: *Similia similibus curantur.*

Among the favorite drugs were antimony, copper, iron and lead. Herbs were used extensively, but most of them are difficult to identify. To mention just a few: the leaves of Koriander, *Kusbarta,* were considered effective on inflamed lids,[217] and *Gargira,* the leaves of white mustard, were recommended by R. Huna for the same condition.[228]

To solve the problem of etiology of disease, in the absence of a clear knowledge of anatomy, pathology and physiology, the Babylonians originated the theory of demonology. Any disease the etiology of which was not clear was attributed to supernatural factors. For example, some contagious diseases were recognized by the Babylonians, but the manner of their spread could not be explained in a natural way; they were therefore ascribed to supernatural sources, such as demons. Superficially, a doctrine like this appears, to say the least, primitive; but if one prefixes the word demon to read microscopic-demon, the theory of demonology, in relation to the practice of medicine, takes on a different aspect. True enough, in the premicroscopic days the demons were invisible, but, nevertheless, they figured out that that invisible object was at the bottom of the transmissible diseases. The Talmud in many respects accepted the demonic theory and made additions to it. R. Yudin, quoting R. Levi, said, "Every inch of space in the air is filled with innumerable demons, ready to seize upon the body and inflict

225. Buck: Medizinische Volksglauben aus Schwaben, Berlin, 1898, p. 44.

226. Heat withdraws heat is an old Talmudic aphorism.

227. Numbers Rabba 18:22. Leviticus Rabba 22. One person became blind from a certain herb; this same herb made a blind one see.

228. Sabbath 109a. The leaves of white mustard were also used by Galen and Pliny in diseases of the eyes (Natural History 20-49). R. Papa claims Gargira to be beneficial to vision. R. Hisdai recommends Tardin (a species of beets) as an ocular tonic (Erubin 29a).

diseases, but fortunately, the Lord has also created angels to protect the afflicted from harm." [229] Here one has a vague hint of pathogenic organisms and antibodies in the disguise of little demons and little angels.

The treatment for demonic diseases was medicine and magic. The medicine was intended to disgust, so to speak, the demons. A genuine remedy was often combined with an unpleasant ingredient, such as turpentine mixed with a green frog, or sesame with doves' dung.

The few quotations and references made illustrate that in spite of the incumbrance of incantation, divination, charms and belief in demons, Biblical and Talmudic medicine has reached the borderlines of medical science. The recognition and description of ocular diseases, the care of the eye and the therapeutic measures taken to combat diseases of the eye deserve a significant share of credit in the development of medicine and hygiene in general and ophthalmology in particular.[230]

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229. Midrash Tankuma, chap. Aikeb, Mishpatim, Shocher Tob, 17-55.

230. To recall only a few minor therapeutic measures: Judaei Emplastrum fracto capiti accommodatum and Judaei compositia are cited by Celsus (De arte medica, lib. 5, chap. 19, no. 2, chap. 22, no. 4), and later by Aetius (Tetrab 4, Sermo 3:14, Ed. Lugdun), and the ad splenem remedium singulare of Patriarch Gamliel is cited by the Roman physician Marcellus Empiricus (De Medicamentis, lib. 34:7. Preuss, Julius: Biblisch-talmudische Medizin, Berlin, S. Karger, 1911, pp. 4 and 6).

LEBER'S DISEASE

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PHILADELPHIA

Beers, in 1817, was probably the first to place on record the history of a case of what would now be regarded as one of Leber's disease. Later, those two clever observers, von Graefe and Jonathan Hutchinson, as well as others, recorded similar histories. But it remained for Leber, then a young man 31 years of age, to type definitely those cases of so-called hereditary or congenital amaurosis and establish what has since been regarded as a definite syndrome. Since this contribution in 1891, but little that is definite has been added to his accurate observations.

In 1904, Gowers stated: "The facts suggest that the inherent vital energy of these structures is inadequate to maintain their nutrition much beyond full development, so that they gradually fail, from abiosis, in what may be designated abiotic atrophy, or abiotrophy." He believed that the onset was determined by some adventitious cause, and, further, what still maintains twenty-eight years later, that "therapeutic measures, when most effective, may only prevent further failure."

Nettleship's Bowman Lecture in 1909 and the full discussion in Wilbrand and Saenger assembled additional data up to the time at which these contributions were made.

In 1912, Bruner described some distortion of the sella in his case of Leber's disease.

In 1916, J. Herbert Fisher called attention to the relationship that the pituitary body may have to this condition and among other observations pointed out that an enlargement of the sella turcica may occur in hereditary optic atrophy. He believed it more rational to assume an inherited tendency to a temporary and transient disorder of the hypophysis than to a similar tendency to involve the papillomacular fibers of the optic nerve.

Writing in 1922, Treacher Collins classified Leber's disease among the ophthalmic abiotrophies. In the Bowman Lecture in 1923, de Schweinitz discussed the possible relations between the condition under discussion and disease of the pituitary body. Now there is available the recent important discussion of this subject by Julia Bell, who

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Read before the Section on Ophthalmology, College of Physicians, Philadelphia, April, 1932.

The visual fields furnished by the author were redrawn in the headquarters office of the Association.

has assembled the data from 240 pedigrees covering 1,182 affected persons. Seventeen of these pedigrees and 164 affected members concerned Japanese families, but the remainder were chiefly of European races.

Bell's observations are important enough to quote freely from them concerning the essential features of this condition. She did not regard the condition as a sex-limited disease and contrasted the male sex incidence among Europeans, 84.8 per cent, with 59.1 among the Japanese. In regard to the onset of the disease, and fully realizing the obvious inaccuracies of observation, she believed it probable that the majority of the cases attained the maximum disability within two months from the time of the apparent onset, and that it was rare for the symptoms to advance after six months. Owing to the variance as to the age of onset in the European and Japanese races, they were cited separately. Thus, among the former the mean age for the male was 23.23 and for the female, 25.15, while in the latter race the mean age for the male was 21.30 as contrasted with 20.50 for the female. While certain figures were produced that seemed to indicate that antedating did occur, a more thorough investigation failed to produce evidence to substantiate this view in passing from one generation to the next.

As to the clinical signs and symptoms, diminution of vision due to a central scotoma was the only symptom complained of in the majority of cases, although this field defect was absent in a small proportion of cases. Where field characteristics were referred to, she found that among 270 males a central scotoma was present in 88.1 per cent, and among 50 females in 82 per cent. Peripheral field contraction had an incidence of 42.5 per cent among 212 males, and 62 per cent among 50 females. Both central scotoma and peripheral contraction were present in 34.6 per cent of 179 males, and in 45.2 per cent of 42 females.

Concerning the disk manifestations, these were found to vary from what might be regarded as a neuritis, to evidences of atrophy at a very early stage, the latter first affecting the temporal sector. Concerning the course and prognosis, nothing was found that indicated that the disease could not run a protracted course, or that it was incompatible with long life.

After citing an incidence of mortality in early childhood in selected pedigrees of 39.1 per cent among affected sib-ships and of 46.6 per cent among unaffected sib-ships, she refrained from any definite statement, but said, "I do not think it would be easy, if possible, to obtain figures showing a similar degree of association from an equal number of pedigrees of other non-fatal hereditary diseases."

As to prognosis among 211 males, 86, or 29 per cent, showed some improvement, 12 of these going on to complete recovery and 15 to marked

improvement, while in the remainder the improvement was less evident. Her statistics seemed to show that when the age of onset was early, i. e., prior to 16 years of age, the ultimate outlook was more favorable. The data were not of such a character as to warrant any prognostic conclusions from the character of the fields. The presence or absence of severe headaches and giddiness, which are sometimes associated in these cases, did not seem to influence the prognosis. From the data presented it was not possible to draw definite conclusions as to the influence of tobacco or alcohol, but she stated that "any habit which tends to lower vitality and powers of resistance must tend to impede recovery in chronic illness whatever its nature may be."

Concerning associated disabilities, she discussed the pituitary gland. Reports of roentgen examinations were available in 60 cases; among these the sella turcica was enlarged in 14 instances. It may be said at this point that Bell did not seem to be enthusiastic about the influence of the pituitary body as an etiologic factor, but on the other hand, regarded the disease under discussion as an abiotrophic disease. As to its hereditary character and its mode of transmission, she stated that she believed there was no doubt that the disease was transmitted generally through the female who was commonly unaffected, but under special conditions the father was able to transmit the disease, either directly or indirectly, through his daughters. Thus, of 573 affected males, 95 per cent were affected through the mother, and of 88 females, 84 per cent through the mother. Finally, she stated that if the sisters of affected males would abstain from parentage the disease would be almost exterminated.

Four case reports are submitted, three of which pertain to the same genealogy. Brief reference is made to a fifth case (fig. 1).

REPORT OF CASES

CASE 1.—F. S., a youth, aged 19, was admitted to the neurosurgical department on July 22, 1929, for supposed pressure on the anterior visual pathways. While in this service his tonsils were removed and the right ethmoid cells were opened. The Wassermann test was negative, and the blood sugar, 85. Roentgen examinations of the base of the skull were negative. No increased pressure of the cerebrospinal fluid was found. The patient was discharged on August 11.

He was readmitted on September 26 to the ophthalmologic service. He stated that he first noted impaired vision in the left eye the latter part of February, 1929; that his attention was first attracted to it while playing baseball, and that on closing the right eye the contrast in the vision of the two eyes was quite marked. Since then vision had become markedly worse. In the latter part of June, the right eye became affected, and as with its fellow the vision rapidly deteriorated. Vision in the right eye was 1/60 eccentric, and in the left eye, 1/200 eccentric. He has not had headache or any ocular discomfort, but stated that he could see better in a subdued light and toward evening. In April, June and July he was studied by three different eye surgeons.

The patient was a tall, slender youth, 6 feet and 3 inches (190.5 cm.) in height. A thorough physical examination gave negative results. Neurologically, his mentality, station and gait, speech and articulation, motor and sensory functions, reflexes and cranial nerves (except second) were all normal.

There were no extra-ocular muscle palsies and no nystagmus; the corneal sensation was intact, and the convergence was poor. When adapted for distance, the right pupil measured 5.5 mm. and the left, 5 mm.; they reacted moderately to direct and indirect light, but poorly on attempts to accommodate and converge. No changes were noted in the media of either eye; the disks showed distinct pallor of the whole temporal portions with large shelving cups. No changes were noted in either macula or in the peripheral portions of the eyegrounds. The retinal veins of the right eye were somewhat larger than those of the left. The form fields were almost full, but there was a large central scotoma in each eye, as shown in figures 2 to 8.

The prostate was normal, as was the spinal fluid. Roentgen examinations of the gallbladder gave negative results. Chemical composition of the blood as regards the urea nitrogen and blood sugar was normal.

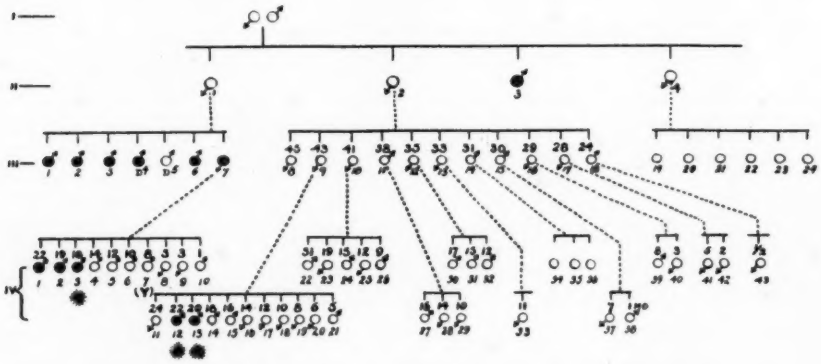

Fig. 1.—In the pedigree the ages are indicated above and the number in each generation below. There was no history of tuberculosis or consanguinity, and one case of epilepsy (*Y*). The individual members examined are indicated by an asterisk. The total number of members in the pedigree was 71; 29 females, 33 males, and 9 sex unknown. The total number supposedly affected was 13: 12 males and 1 female. Among 40 members in the fourth generation whose ages were known, 28 children were under 16 years of age and 12 were older. Of the 12 older children, 5 were affected, all males.

On December 12, the ocular conditions were much the same, except that the scotomas were larger. Vision in the right eye was 1/300; with the left eye, fingers could be counted at 6 inches.

On May 27, 1930, vision in the right eye was 1/200; the left eye, 1/120; on June 20, it was 1/100 in the right eye and 1/45 in the left, and on April 14, 1932, it was 3/100 in the right eye and 3/100 in the left.

CASE 2.—S. S., a youth, aged 19, was first seen at the University Hospital on Oct. 15, 1931, and was admitted to the ward for study on October 26. He was a brother of the patient in case 1. He first noted failing vision in the right eye two months previously. Shortly after this the left eye became similarly affected, and photophobia developed. This was followed by some vague pain in the eyes, which

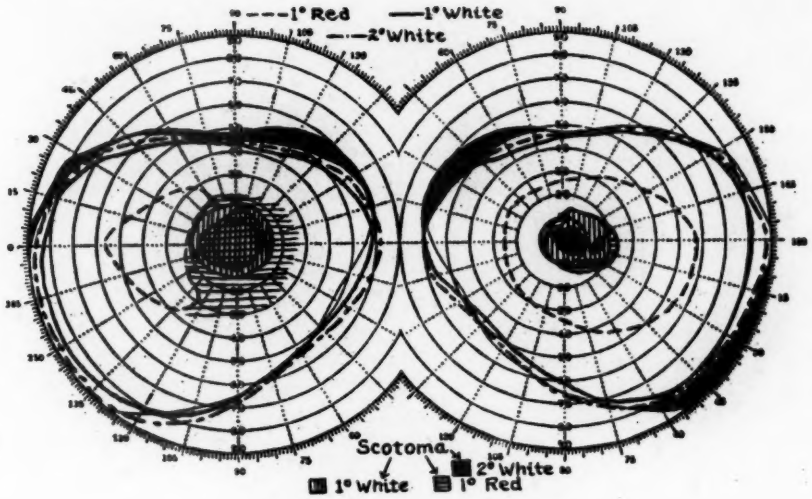

Fig. 2—F. S., July 23, 1929.

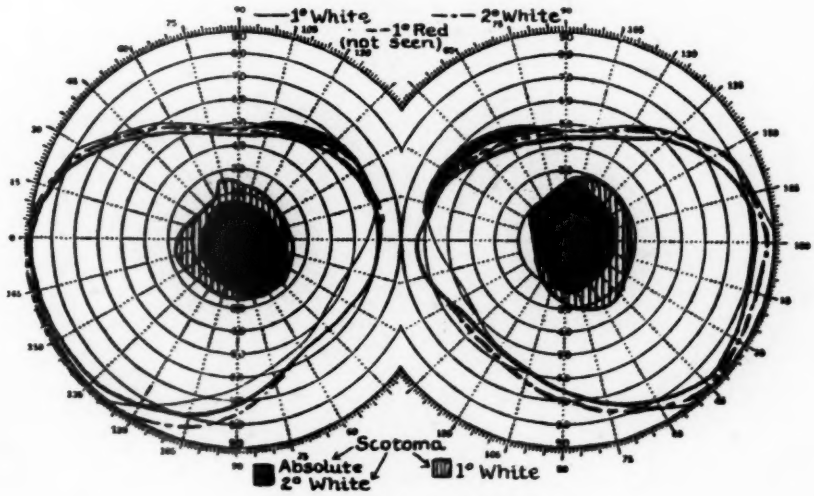

Fig. 3—F. S., Nov. 15, 1929.

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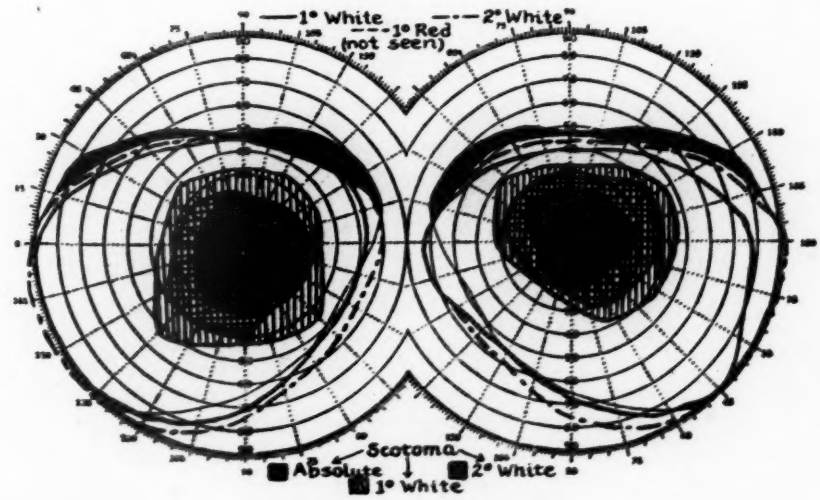

Fig. 4—F. S., Nov. 29, 1929.

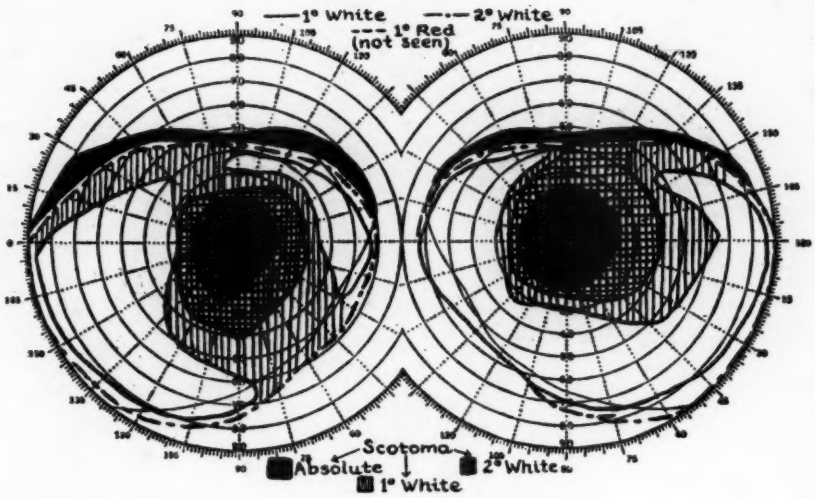

Fig. 5—F. S., Dec. 9, 1929.

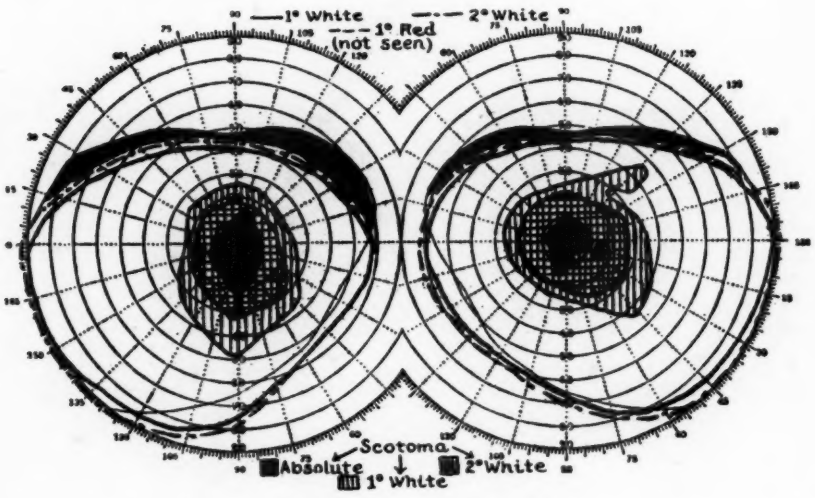

Fig. 6.—F. S., Feb. 24, 1930.

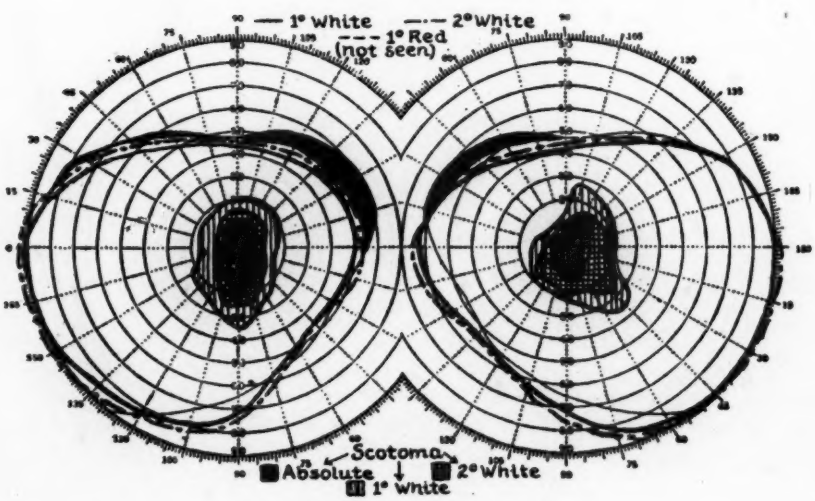

Fig. 7.—F. S., March 4, 1930.

persisted but a few days. From the time of onset to the present there has been some vague headache. On admission his only complaint concerned his vision, and he described the defect as a black spot which seemed to cover anything he looked at.

His mother, aged 38 years, was suffering from some indefinite nervous disease. His father, aged 48, was well. He had six brothers and five sisters, all living and well. The oldest child was the patient in case 1.

The patient was a farmhand and did not use alcohol, tobacco or drugs. As with his brother, there was no history of preceding night blindness. The general physical findings quite confirmed the absence of symptoms, for no pathologic signs were found that were referable to the neck, chest, heart, abdomen or pelvis.

Roentgen examination revealed several abscessed teeth, and these were removed in the general clean-up of the mouth. A similar examination of the accessory sinuses gave negative results and this was confirmed by clinical observation. The

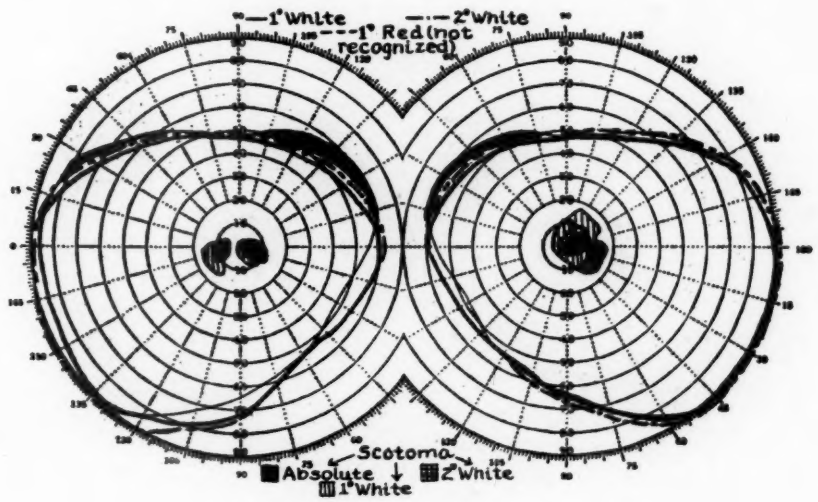

Fig. 8—F. S., April 14, 1932.

tonsils were septic and were removed. Roentgen examination of the head was negative except for an enlarged pituitary fossa; the anteroposterior measurement was 13 mm., and the depth, 10 mm.[1] The prostate was normal. The basal metabolism was + 3.

The blood count and differential count were normal. The Wassermann test was negative. The urea nitrogen and blood sugar content were normal. The urine was normal.

Externally, each eye was free from manifestations of disease. On each side the orbicularis was intact, as was the corneal sensation. There was no nystagmus or

1. Dr. Pancoast and his associates in the department of radiology at the University Hospital regard any measurement of the sella turcica exceeding 12 mm., anteroposteriorly and 10 mm. in depth as indicating an enlargement. The first measurement represents the greatest anteroposterior measurement of the fossa, the latter the greatest depth as measured from the diaphragma sellae. Kornblum, Karl: Alterations in the Structure of the Sella Turcica, Arch. Neurol. & Psychol. **27**:305 (Feb.) 1932.

ocular palsies. The pupils were of average size and equal, and reacted moderately to the various reflexes. Intra-ocularly, the media were clear; the disks showed an evident pallor of the temporal sectors with quite good capillarity elsewhere, and in the left eye a superficial capillarity remained even over the temporal sector. There were no macular or peripheral lesions of the choroid or retina. The retinal vessels were about a minimum normal in caliber. The fields were as shown in figure 9. At the time of admission the patient could count fingers at 1 meter with each eye. On April 14, 1932, vision in the right eye was 3/100; in the left eye, 3/100.

Case 3.—J. R., a youth, aged 18, was a cousin of the patients in cases 1 and 2. The grandmothers were sisters. The mother of this patient was similarly affected. He never had any ocular diseases until the early part of January, 1930, when he noted that he could not see the writing on the blackboard when in school. His vision became gradually worse, and he stopped school in February, 1930. The vision reached its maximum impairment in March.

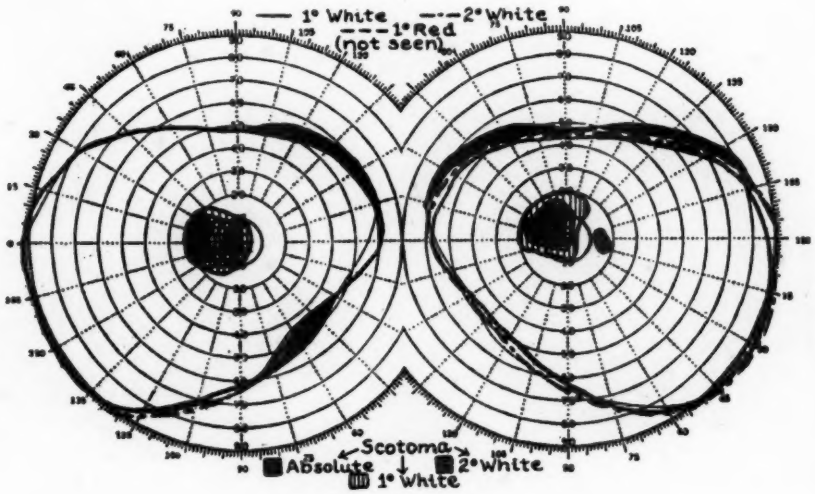

Fig. 9—S. S., Oct. 27, 1931.

At the present time he can read the headlines in the newspapers with great effort. He never had any severe headaches or dizziness, but at times had slight ocular discomfort after persistent efforts in reading. He had never noted any night blindness, either before or since the onset of his visual impairment. Several doctors had been consulted, but he was advised that nothing could be done for him. There were no symptoms referable to the cerebrospinal system, ears, nose and throat, chest, cardiovasculorenal system or genito-urinary tract.

He stated he had "encephalitis" when 5 years of age and was ill for one week. He had had whooping cough and measles. His tonsils were removed in 1931. He does not use alcohol, but smokes a package of cigarets every two days. Since stopping school, he has worked in his father's cider mill.

Examination of the eyes showed that the palpebral fissures were equal and the orbicularis intact. The rotations were full, and convergence was slight. The pupils reacted moderately to direct and indirect light, and only slightly to attempts at convergence and accommodation. The corneal sensation was intact. In each eye the disk revealed a moderate atrophic pallor of the temporal sector, but a

dusky capillarity elsewhere, which might be regarded as the gray-red type. The caliber of the vessels was within normal limits. There were no macular or peripheral lesions. Vision in the right eye was 6/100; in the left eye, 6/100 (fig. 10).

CASE 4.—P. R., a man, aged 34, was first seen Dec. 23, 1914. In September, 1907, he first noted that he could not see the markings on an engineer's scale with the right eye. The following January or February, he noted impairment of vision in the left eye, and the deterioration was so rapid that he was practically blind within a week. By that time the right eye had failed to a similar extent. From the age of 18 to the time of onset of his ocular trouble he was subject to severe "neuralgic headaches" associated with pain through the eyes, which developed every three to six weeks.

The patient stated that in the last four generations, on his mother's side one male had been similarly affected, and in each instance through an unaffected

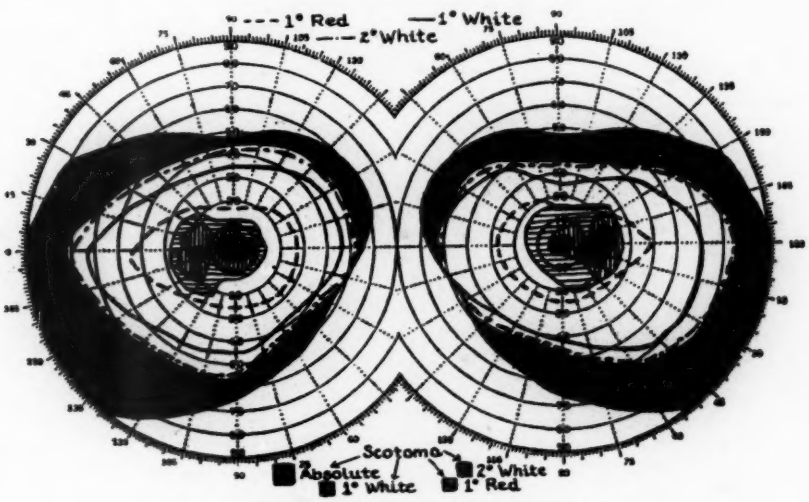

Fig. 10—J. R., April 14, 1932.

female. An uncle with the same condition had advised him that all were affected before the age of 28 and each one a few years after marriage.

The patient had had influenza a number of times and jaundice at the age of 4 years. He had had several attacks of tonsillitis. No complete physical examination could be made at that time, but he did have a sinusitis.

Examination of the eyes showed that the orbicularis was intact, as was the corneal sensation. The rotations were full; there was no convergence. The pupils were equal and reacted to direct and indirect light. In each eye the media were clear, the disks well defined, atrophic and of bluish-white color; there were no lesions of the choroid or retina, and the retinal vessels were of normal caliber. The fields were as shown in figure 11. Vision in the right eye was 4/150; in the left eye, 4/150.

When the patient was seen again in 1918 for a trifling condition of the lid, the vision of the right eye had remained unchanged; vision in the left eye was 3/150. In January, 1918, he had a submucus resection, but still had evidences of a chronic sinusitis; the tonsils had not been removed. There were no abscessed teeth. No pathologic lesions were found in the lungs, heart or abdominal organs.

The blood pressure was 138 systolic and 100 diastolic; the Wassermann reaction of the serum was negative. The urine was normal. Prior to 1914, while under the care of an oculist, he took 90 drops of nux vomica three times a day.

For twenty years he had used alcohol continuously and had smoked from three to four cigars and a package of cigarets a day.

The father died eighteen years ago of cholecystitis; the mother was living and free from the condition affecting the patient. Two brothers died in infancy. (Dr. A. C. Morgan supplied the later medical notes.)

Case 1 illustrates the importance of the realization that the medical side of ophthalmology is just as important as the surgical side, although one would not realize this on a visit to the ophthalmic wards of most of the hospitals. A patient with a squint of years' standing seems to have

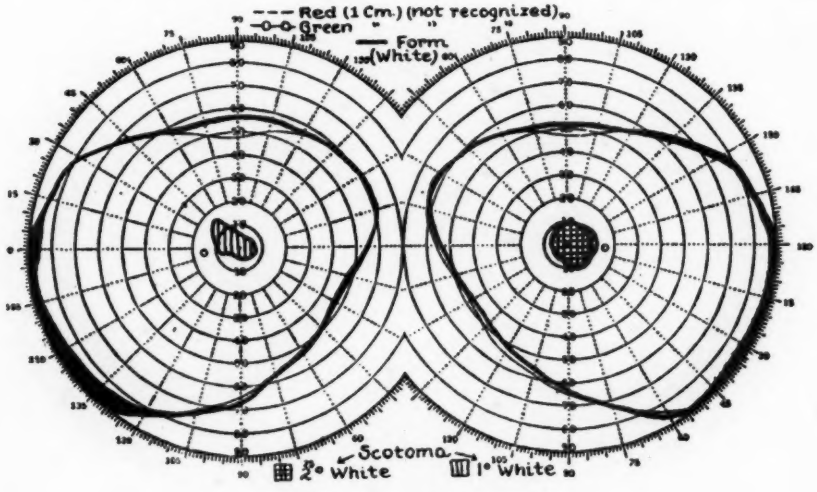

Fig. 11.—P. R., Dec. 31, 1914.

a much better opportunity for a bed than one with a partial optic atrophy or a uveitis in the absence of acute anterior uveal involvement. Despite repeated questioning as to consanguinity and heredity in the first case, no positive information could be obtained until after the patient's younger brother became affected eighteen months later. It was not until March, 1932, that we obtained data concerning the relatives, and but a week ago the details of the pedigree. If contact had been lost with the first patient, his case would have been regarded as one of retrobulbar neuritis due to focal infection, i. e., of the accessory sinuses and tonsils. If no focus had been found, some enthusiasts concerning multiple sclerosis probably would have anticipated that condition which, in my opinion, would have been thoroughly unjustifiable. This pedigree also emphasizes the importance of a follow-up system.

Another interesting point in connection with this disease is the little consideration given to Leber's disease when discussing the possible

lesions about the chiasm. Given a patient with a field such as existed in case 1 on Dec. 9, 1929, and associate with it an enlarged pituitary fossa such as was present in the case of the patient's brother, would such a patient have been operated on by the average neurosurgeon, and if so, would it have been justifiable? Now, fortunately, this is not a common disease, and unfortunately an accurate family history is sometimes lacking, but under any circumstances, I have not the least doubt that an occasional patient may have come to operation without there being a full knowledge of the underlying etiologic facts.

Of course, certain patients have been subjected to operation when the underlying condition was fully recognized. Only recently, Kuhn [2] advised an operation on his patient in the absence of pituitary signs or symptoms and when roentgen examination revealed a small sella turcica. To illustrate further the occasional difficulties that at times attend the diagnosis of Leber's disease and also the importance of careful and painstaking case histories, I recently saw a case that had passed through the hands of three of the highest type of clinicians, but the probably proper diagnosis was not made until twenty-two years after the patient was first observed.

Certainly no case history concerning optic atrophy is complete unless it includes some statement as to consanguinity, heredity and industrial exposures, in addition to the usual information as to the past medical history and social habits of the patient. This naturally brings up the question of the relationship of Leber's disease to industrial pursuits, especially in these days of compensation laws. Given a young man, 22 years of age and a member of an afflicted family, who while working in a chemical plant exposed to noxious fumes developed a retrobulbar neuritis. Should he receive compensation? An example analogous to this occurred in the present pedigree and compensation was paid. Doubtless an hereditary taint or disease was not suspected, and probably no inquiries were made concerning it.

Despite the fact that this disease has been recognized for over a hundred years, no pathologic report concerning it was available until the past year, when Rehsteiner [3] recorded the findings seven years after the onset of the disease. The only intra-ocular changes were atrophy of the ganglion cells and of the nerve fiber layer. While some parts of the optic nerve were normal, other portions were markedly atrophic and contained but few or no medullary sheaths. The atrophic portion corresponded to the position of the papillomacular fibers. Here there was

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2. Kuhn, H. S.: Hereditary Optic Atrophy (Leber's Disease), Arch. Ophth. **5**:408 (March) 1931.

3. Rehsteiner, Karl: Arch. f. Ophth. **125**:14, 1930; abstr., Brit. J. Ophth. **16**:241, 1932.

an increase in the glia fibers associated with an atrophy of the finer connective tissue septums. No signs of inflammation were present. After commenting on the similarity of the nerve findings to those seen in atoxyl poisoning, these findings being in contrast to the thickened septums found in postneuritic atrophy, Rehsteiner concluded that inflammation was absent even in the early stages and that the disease was dependent on an inherited primary degeneration. He did not regard the findings as significant of a toxic origin from endocrine disturbance.

ANTERIOR CHAMBER PUNCTURES IN RELATION TO INTRA-OCULAR TENSION

PETER C. KRONFELD, M.D.
CHICAGO

I appreciate greatly Dr. Abraham's [1] attempt to clarify a subject which to me—despite a great deal of attention and work on my part—is still full of unsettled questions and problems. In his paper on anterior chamber punctures, Dr. Abraham gives a sketchy survey of the literature concerning regeneration of the aqueous. He might have added the conclusion of the most recent reviewer of this subject, Baurmann,[2] who stated the difference between the reaction of human and that of animal eyes to withdrawal of the aqueous is "jedenfalls sehr markant" (at all events, very marked). Dr. Abraham also might have quoted me [3] as one of the supporters of Wessely's theory that the difference between the regeneration of the aqueous in the eyes of human beings and animals is only a quantitative one, which theory Dr. Abraham seems to accept. Since the subject of our controversy is the existence or nonexistence of a quantitative difference between the normal and the glaucomatous human eye in its response to the emptying of the anterior chamber, it is obvious that data obtained from animal experiments are here of little value.

Dr. Abraham [4] stated a theory of his own, the nucleus of which is as follows: The degree of the reactive hypertension that follows the puncture of the anterior chamber parallels the visual function of the eye. An amblyopic eye gives a less marked reaction than an eye with normal vision.

Dr. Abraham based this theory mainly on his findings in a man aged 25 (his case 4), whose left amblyopic, highly myopic eye gave a much lower tension curve during the first two and a half hours following the puncture than the normal mate. Dr. Abraham apparently overlooked the fact that the volume of the anterior chamber was very dif-

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From the Eye Clinic of the University of Chicago; Dr. E. V. L. Brown, Director.

1. Abraham, S.: Anterior Chamber Punctures in Relation to Intra-Ocular Tension: A Critical Study. Arch. Ophth. **7**:888 (June) 1932.

2. Baurmann, M.: Die Photorezeptoren, in Bethe, von Bergmann, Embden and Ellinger: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1931, vol. 2, p. 1324.

3. Kronfeld, P. C.: Intra-Ocular Tension Following Puncture of Anterior Chamber, Arch. Ophth. **1**:458 (April) 1929.

4. Abraham,[1] p. 893.

ferent in the two eyes (0.2 cc. in the right; 0.29 cc. in the left). Suppose one assumes that the ciliary body of the myopic eye produces the "second" aqueous at the *same* rate as that of the normal eye. This assumption is in Dr. Abraham's favor, but is not probable if one considers the well known anatomic differences between the shape of the ciliary body in myopic and in normal eyes. If these differences are ignored for the time being, it is easily understood that the rise of tension in the myopic eye cannot keep up with that in the normal eye because the latter requires only two thirds of the amount of fluid necessary in the former eye for the restoration of the anterior chamber. The importance of the volume of the anterior chamber in relation to the

TABLE 1.—*Correlation Between Volume of Anterior Chamber and Abruptness of Tension Curve in Six Cases*

Cases arranged according to abruptness of tension curve after anterior chamber puncture	Case 5	Case 4	Case 6	Case 2	Case 1	Case 3
Respective volumes of anterior chamber, cc	0.18	0.20	0.22	0.22	0.26	0.30

TABLE 2.—*Data on Five Nonglaucomatous Eyes*

Case	Age	Optic Nerve	Vision
1	45	Partial optic atrophy; questionable hypopituitarism	0.5-0.6
2	70	Normal; mild retinal arteriosclerosis	1.2
3	29	Normal	0.3 amblyopia ex anopsia
4	44	Partial atrophy; chronic alcoholism	0.8
5	10	Normal	0.8-3 alternating strabismus

abruptness of the tension curve is easily seen in table 1, in which six cases of senile cataract are tabulated in regard to those two factors (see also chart 3).

The two factors are easily seen to be inversely proportional. The six cases will be given later in detail. They certainly prove that the steepness or abruptness of the tension curve following puncture of the anterior chamber depends, *ceteris paribus*, on the volume of the anterior chamber.

If Dr. Abraham wants to prove his theory he will have to compare two eyes of a patient whose anterior chambers have approximately the same volume.

The tension curve obtained from the normal eye of Dr. Abraham's patient resembles closely that of Magitot's patient;[4a] the fact that Magitot's patient had an optic atrophy, while Dr. Abraham's patient had a normal eye, does not speak in favor of the theory of the latter author.

4a. Kronfeld,[3] p. 450.

Dr. Abraham is thoroughly mistaken if he believes that my "normal" cases speak in favor of his theory that amblyopia leads to a low tension curve after the puncture of the anterior chamber. In the paper which I read before the New York Academy of Medicine (Dec. 15, 1930), I presented the tension curves of five nonglaucomatous eyes which Dr. Abraham must have known quite well. The curves are shown in chart 1 and the other data in table 2.

In these findings I can see nothing that supports Dr. Abraham's theory. The curve of the truly amblyopic eye (case 3) is not essentially different from that of the normal eye (case 2).

The effect of atropine on the tension curve after the puncture, which Dr. Abraham referred to in his paper,[5] was first demonstrated by Magitot [6] and then confirmed by me in the case of an eye of a child.

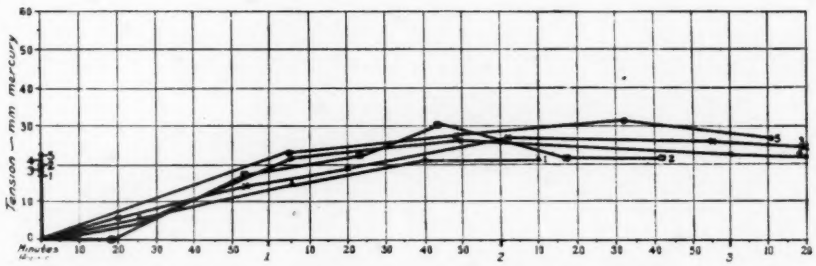

Chart 1.—Tension curves following anterior chamber puncture in five nonglaucomatous eyes. The other observations are given in table 2.

E. O., a girl, aged 10 years, first seen on June 18, 1930, had convergent alternating strabismus of 30 degrees. Examination (under atropine) showed: V.R. +1.00 +0.25 cyl. ax. 90 = 0.8 + 3; V.L. + 1.00 + 0.50 cyl. ax. = 0.8 + 3. On June 21 a left internus rectus recession was done, which corrected most of the convergence.

Five months later two anterior chamber punctures were performed on the left eye, the first one without any previous medication, the second one three weeks later under the influence of atropine (chart 2). Dr. Abraham must have known of this case because I presented it before a staff conference early in December, 1930.

My case 6, quoted by Dr. Abraham (his chart 5), does not necessarily prove the restraining influence of atropine on the reactive hypertension because at the time of the first puncture both anterior chambers were emptied within a few minutes, which may have intensified the reactive hypertonia. The effect of epinephrine on the reaction of a human eye to anterior chamber puncture was also shown by Magitot [6] and by me.[3] This drug evidently hampers the reactive hypertonia following the paracentesis.

5. Abraham,[1] p. 894.
6. Magitot, A.: Ann. d'ocul. **160**:165 (March) 1923.

Before I consider the cases reported by Dr. Abraham I should like to mention some further attempts which I made to detect a possible difference between an eye affected with primary simple glaucoma and a nonglaucomatous eye which in every other respect was comparable to the glaucomatous eye. That meant that only eyes of aged people could be considered. I could have used some of the "normal" cases reported in my paper in the *Zeitschrift für Augenheilkunde*.[7] Unfortunately I found that the three tonometers which I used for the tensions on those patients gave incongruous readings when used on a rubber model. It finally came to my mind that patients with senile cataract could well be used for this purpose because incipient cataract is a frequent finding in glaucomatous eyes. Chart 3 gives the results obtained in six consecutive patients with senile cataract who were admitted to Billings Hospital of the University of Chicago for operation. In none of these six

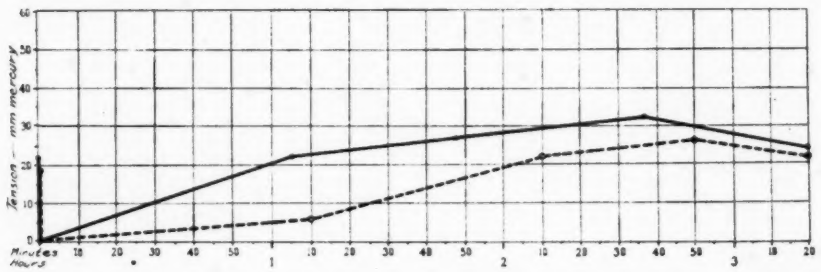

Chart 2.—Curves for E. O., a girl, aged 10 years. The anterior chamber of the left eye was punctured twice, the first time without previous medication (continuous line); the second time three weeks later, under the influence of atropine (dotted line). The respective volumes of aqueous obtained were 0.27 and 0.26 cc.

cases did the postoperative course and subsequent examination suggest the presence of glaucoma. In each case I performed the puncture and emptied the chamber completely, which fact was demonstrated by a jerky contraction of the pupil (as mentioned in my German paper [7]) and by a slight groove in the iris produced by the needle. In every one of the punctures performed *lege artis*, the needle came in blunt contact with the iris. From my experience I do not believe that this itself adds to the reactive hypertonia in human eyes.

REPORT OF CASES

Case 1.—Mrs. L. M., aged 56, had a nuclear cataract in the right eye and an incipient cataract in the left. Vision in the right eye amounted to perception and projection of light. The right anterior chamber was emptied on Jan. 12, 1932. The volume was found to be 0.26 cc. On January 13, extraction of the cataract in the right eye was performed without complication.

7. Kronfeld, P. C.: Ztschr. f. Augenh. **71**:58 (March) 1930.

CASE 2.—Mr. H. R., aged 62, had cataracta nigra in each eye. In the right eye vision was 3/200; in the left, 0.3. On Jan. 28, 1932, the right anterior chamber was evacuated (volume, 0.22 cc.), and on the following day the right cataract was extracted. The operation was followed by a mild postoperative iridocyclitis, which subsided under fever and local treatment.

CASE 3.—Mr. C. B., aged 65, had cataracta nigra in each eye. In the right eye vision was perception of fingers at 3 feet (91 cm.); in the left eye, 0.1. On Feb. 1, 1932, the right anterior chamber was punctured (volume, 0.30 cc.), and on the following day the lens extraction was performed. There was no complication.

CASE 4.—Mrs. J. J., aged 49, had an incipient cataract in the right eye and a mature cataract in the left. With the right eye she perceived fingers at 3 feet; in the left eye, at 1 foot. An anterior chamber puncture was performed on the left eye Feb. 11, 1932 (volume, 0.20 cc.); a lens extraction, on February 12.

CASE 5.—Mrs. M. W., aged 62, showed a mature cataract in the right eye and an incipient cataract in the left. An anterior chamber puncture was performed on the right eye on Feb. 16, 1932 (volume, 0.18 cc.), and a lens extraction on February

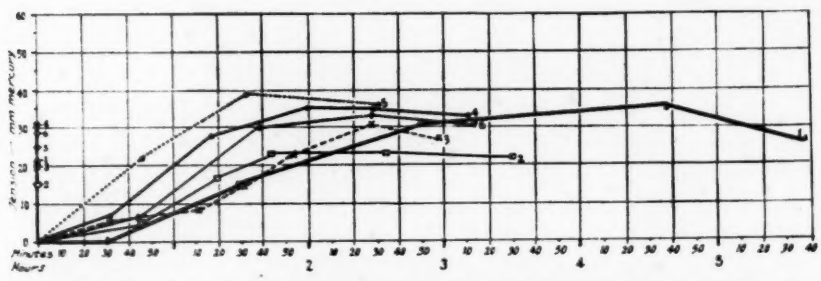

Chart 3.—Tension curves following anterior chamber puncture in six cases of senile cataract. The other observations are given in the text.

17. Some vitreous was lost after the expression of the nucleus (the patient was a "squeezer").

CASE 6.—Mrs. C. J., aged 60, had an incipient cataract in each eye. In the right eye the vision amounted to perception of fingers at 2 feet (61 cm.), and in the left eye to 4/200. An anterior chamber puncture was performed on the right eye on Feb. 23, 1932 (volume, 0.22 cc.), and an uncomplicated lens extraction on February 24.

The curves shown in chart 3 lie, as a whole, a little higher than those obtained from normal eyes of younger people, most likely because of senile rigidity of the sclerotic and of senile vascular changes, both of which have to be expected in the large majority of eyes with primary simple glaucoma. To me it seems as though the cases of chart 3 approached the standard of the nonglaucomatous eye of people over 50 years of age as closely as possible at this time. At least, I have not had the opportunity of suggesting or performing anterior chamber punctures on eyes more suitable for the purpose of setting up a standard for comparison.

To the discussion of the nine cases reported by Dr. Abraham, I wish to add the following comment:

In his case 1 (M. F., a woman, aged 67) Dr. Abraham's summary was:

> In this case there is no reason to consider glaucoma if one ignores the data furnished by anterior chamber puncture. Sclerosis of the lenses rather than glaucoma explains the decreased vision and the almost constant colours around lights.

I have never seen this patient, but from the clinical records and the reports sent to me by other ophthalmologists I believe that there is a definite reason for considering glaucoma even if the data furnished by the anterior chamber puncture are ignored. I wish to draw the attention of the reader to the following abstract of the patient's record, which contains mainly the tensions found at the various examinations. If not stated otherwise they were taken with the Schiötz tonometer and the readings transposed into millimeters of mercury according to the new scale. The figures in parentheses give the diameter of the pupil in millimeters.

March 31, 1927: Patient under miotic. R.T. = 30; L.T. = 27 (McLean direct reading instrument, Dr. W. H. Roberts, Pasadena, Calif.).

Jan. 30, 1928: R.T. = 32-34; L.T. = 40-42 (McLean tonometer). One per cent pilocarpine ordered.

February 8: R.T. = 35; L.T. = 40 (McLean tonometer).

February 15: R.T. = 30; L.T. = 41 (McLean tonometer).

March 1: R.T. = 31; L.T. = 39 (McLean tonometer).

March 14: R.T. = 35; L.T. = 40 (McLean tonometer).

May, 1928, to July, 1930: Tension of both eyes normal (recorded by Dr. T. Allen, of Chicago), except after dilatation of the pupils with euphthalmin, when almost always there was an increase of tension of several points, at one time reaching 40 mm. of mercury (Schiötz).

July 19, 1930: Patient first seen at the University of Chicago Eye Clinic. R.T. = 29; L.T. = 29.

July 23: R.T = 23 (2.5); L.T. = 29 (2.5).

July 25: R.T. = 22 (2.5); L.T. = 29 (2.5).

July 30: R.T. = 23 (1.5); L.T. = 25 (1.5). Homatropine was instilled in both eyes six times. R.T. = 23 (4.5); L.T. = 29 (4.5).

August 5: Anterior chamber puncture. One per cent pilocarpine ordered.

August 7: R.T. = 22 (1.5); L.T. = 23 (1.5). Pilocarpine continued.

August 18: Under pilocarpine, R.T. = 23 (1.0); L.T. = 20 (1.25).

August 26: Under pilocarpine, R.T. = 22 (1.5); L.T. = 20 (1.5).

November 17: Under pilocarpine, R.T. = 22 (2.0); L.T. = 23 (2.0).

December 23: Under pilocarpine, R.T. = 28; L.T. = 31 (McLean tonometer, Dr. W. Roberts).

December 29: After not having used pilocarpine the previous night or that morning, R.T. = 32; L.T. = 37 (McLean). One per cent pilocarpine continued.

Feb. 24, 1931: R.T. = 31; L.T. = 37 (McLean).

March 27: R.T. = 31; L.T. = 37 (McLean).

June 27: Still under pilocarpine, R.T. = 22 (1.5); L.T. = 23 (1.5). Pilocarpine discontinued.

June 29: R.T. = 23 (3.5); L.T. = 31 (3.5). Homatropine instilled in both eyes six times. Pupils dilated to 4.5 mm. R.T. = 23; L.T. = 26-29. Atropine ordered.

June 30: Under atropine, R.T. = 27-28 (4.5); L.T. = 31 (4.5). Pilocarpine ordered again.

August 20: Had not used pilocarpine for two weeks. R.T. = 27 (2.25); L.T. = 31 (2.25). Pilocarpine, 2 per cent, ordered.

September 1: R.T. = 22 (1.5); L.T. = 39 (1.9). Pilocarpine continued.

September 10: R.T. = 25 (1.75); L.T. 20 (1.75). Pilcarpine continued.

October 20: R.T. = 23 (1.5); L.T. = 25 (1.5). Pilocarpine continued.

December 9: Under miotic, R.T. = 31; L.T. = 33 (McLean) (recorded by Dr. W. Roberts). Pilocarpine continued.

Feb. 17, 1932: Under pilocarpine, R.T. = 31; L.T. = 36 (McLean).

May 9: Under pilocarpine, R.T. = 23 (2.0); L.T. = 31 (2.0).

June 11: Under pilocarpine, R.T. = 25 (1.0); L.T. = 23 (1.0).

July 9: R.T. = 20 (2.0); L.T. = 23 (1.5).

Sept. 19: R.T. = 27 (2.0); L.T. = 31 (1.5).

Dr. Abraham's statement about the patient's visual fields is not correct. Her isopters for 1 degree red were always found decidedly contracted (down to a radius of 20 degrees and less), which suggests strongly the presence of fine arcuate paracentral scotomas. The last central fields were taken on Sept. 19, 1932, and were found normal for 1 degree white and red targets, which also does not exclude the presence of a Seidel scotoma. The disks were described as having shallow excavations which reached the edge over one half of the circumference. The anterior chamber was not emptied completely when the puncture was performed (note the tension of 7 immediately after withdrawal of the fluid). The rise of tension probably would have been still more prompt if the chamber had been completely evacuated.

The left tension was consistently high normal and on various occasions distinctly above the normal limits. Mydriatics had a definite tendency to increase the tension still further. Tensions were never taken during the night. The condition of the optic nerves and fields was at least questionable, certainly far from normal. Should one not consider glaucoma in this case?

Dr. Abraham's case 2 (C. T., a woman, aged 51) was a borderline one which I never intended to use to prove the correctness or incorrectness of my idea concerning the puncture of the anterior chamber. As a matter of fact, I had to classify the case as one not showing simple glaucoma from the result of the repeated anterior chamber puncture (no tension over 42). The reasons why I still am inclined to assume a very early stage of glaucoma in this case are the consistent high normal tensions and a beginning Seidel scotoma in one eye, which was only found once and then disappeared again. It may be a matter of opinion whether these cases showing high normal tensions should be classified as potential glaucomas or not. In this instance I have been unable to arrive at any definite conclusion. The patient's subjective complaints may well be caused by a muscular disorder, as Dr. Abraham believes.

I thoroughly agree with Dr. Abraham in that his case 3 (Z. P., a woman, aged 35) evidently presented no simple primary glaucoma. This diagnosis apparently was a mistake made under the influence of the two high tensions in the left eye (42 and 36) observed after admission to the hospital. The tension curve after the anterior chamber puncture was a little high but not very different from that of a normal eye. On Aug. 15, 1931, I made the following note in the patient's clinical record:

> The high tension may have been produced by an episcleritis or tenonitis which was just fading away when the patient was admitted to the hospital or they may have been artificial throughout. The most logical explanation seems to be: temporary upset of tension because of recurrent episcleritis. A vasomotor imbalance probably produced the high curve. The diagnosis of glaucoma simplex does not seem justified.

From Dr. Abraham's description of his cases 1 and 3 I get the impression that he believes that no increase in tension after the instillation of atropine excludes primary simple glaucoma. This is not true, as one can easily learn from many references in the literature, among which I should like to mention Thiel [8] and Schweigger.[9]

Case 4 in Dr. Abraham's series (J. J., a man, aged 25) has already been discussed.

Cases 5 and 6 were in patients with secondary glaucoma. I am not certain as to what Dr. Abraham wants to prove with these two cases.

It looks to me as though he really did not know what I stated in regard to the difference between primary and secondary glaucoma in their response to anterior chamber puncture. I believe that I expressed myself very clearly in the discussion of my paper read before the New York Academy of Medicine:[10]

> . . . my . . . differentiation [based on the result of the anterior chamber puncture] was between eyes (group 1) showing glaucoma due to retention and eyes (group 2) in which the glaucoma was not caused by retention alone. In the first group there are some cases of primary glaucoma, certain types of secondary glaucoma and possibly the secondary glaucoma that occurs in cases of thrombosis of the central vein. In group 2, the disturbance in inflow, in my opinion, seems to be the leading factor, and in this group I should like to include most, but not all, of the primary glaucomas. In the same group I found a certain number of secondary glaucomas, especially of the type in which the glaucoma comes on with the first attack of cyclitis. So you see, this differentiation does not coincide with the differentiation between primary and secondary glaucoma.

Dr. Abraham's case 5 resembles very much case 9 in my publication [7] in 1930 and should probably be interpreted as mine was, namely, as

8. Thiel, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 4, p. 711.

9. Schweigger, quoted by Peter: Glaucoma, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 3, Leipzig, Wilhelm Engelmann, p. 151.

10. Kronfeld, P. C.: Puncture of the Anterior Chamber in Glaucoma, Arch. Ophth. **5**:674 (April) 1931.

pure retention glaucoma. Dr. Abraham's case 6 is very similar to my case 8.[7] They apparently represent the two most common types of secondary glaucoma.

Cases 7 and 9 of Dr. Abraham's have in common that the anterior chamber puncture was done with a faulty technic. In case 7 the anterior chamber was not emptied completely, which fact was evidenced by the tension (13) immediately after the puncture.

The effect of incomplete emptying of the anterior chamber in glaucomatous eyes has been the subject of special studies on my part, the results of which will be published at a later date. At any rate, I believe I have the right to request that criticisms of my work be based on experiments done with exactly the same technic that I am using. Besides, there is another reason which makes me believe that no conclusion can be drawn from the tension curve of case 7 because it suggests strongly a leakage from the anterior chamber. The tension went up first, but

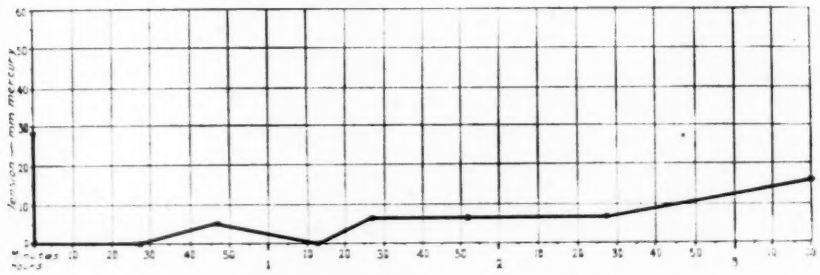

Chart 4.—The correct tensions in Dr. Abraham's case 9.

returned to 13 two hours after the puncture, which occurred in no other case of mine (not even in any with a low normal tension). The reactive hypertonia never subsides to a tension of 13 within two hours.

A similar mistake must have been made when the puncture was performed in case 9 of Dr. Abraham's series. Forty-seven minutes after the puncture the tension was up to 5; twenty-five minutes later it was found to be 0 again—a cleancut case of fistulization. The reason why I pronounced the results of the anterior chamber puncture absolutely negative was that it did not furnish any information because of faulty technic. In addition to the technical mistake made at the time of the puncture, Dr. Abraham made a mistake when he drew the tension curve as he gives it in his chart 8C. The correct findings are given in chart 4 of this paper.

An anterior chamber puncture cannot furnish reliable results if the new-formed aqueous can escape through the opening made with the needle. In case 9 it must therefore be said that one does not know how the patient's eye would have responded to an anterior chamber puncture done *lege artis*.

Concerning case 8 may I remind Dr. Abraham that the tonometer readings of 56 before the puncture and 52 after the puncture do not justify his statement that the tension did not reach the original height?

Furthermore, I should like to remind him of a statement that I made in my German paper,[11] concerning the three phases of the tension curve following puncture of the anterior chamber: (1) the return of the tension to its original height, (2) the reactive, relative hypertonia and (3) the reactive hypotonia.

The first phase seemed to be very regular in the cases of simple glaucoma. "Phases 2 and 3 were not so regular. . . . The majority of eyes affected with glaucoma simplex showed a definite phase 2 . . .", but not all of them. In the same paper [12] I also mentioned the "Inkonstanz" of phase 2. Dr. Abraham's case 8 could be one of those cases in which, after the puncture, the tension did not exceed the original level if the difference in the tonometer readings had been greater than the limit of error in tonometry.

Dr. Abraham's statement [13] that the information furnished by an anterior chamber puncture could more easily be obtained by massaging the eye may be correct. I suggested this possibility in my paper in the *Zeitschrift für Augenheilkunde*.[14] The close similarity of anterior chamber punctures and massage in their effect on intra-ocular tension was recently stressed again by Magitot.[15]

Dr. Abraham's conclusion is: "At present there are no acceptable data showing that the tension after anterior chamber puncture is consistently higher or appears consistently more quickly in glaucomatous eyes" than in the nonglaucomatous eye—*ceteris paribus*.

Dr. Abraham bases his conclusions on his observations in six patients —if his normal eyes are omitted. In two of these faulty technic was patently evident. Two other patients obviously had secondary glaucoma, in which reactive hypertonia beyond that of the nonglaucomatous eye is by no means characteristic, as I have noted on several occasions. The remaining two cases behaved typically, as they showed a definitely abnormal degree of reactive hypertonia. Both patients, in my opinion and in that of other ophthalmologists, had some form of primary glaucoma.

No further discussion of this subject would therefore seem necessary if it were only my intention to reply to Dr. Abraham's criticisms.

11. Kronfeld,[7] p. 53.
12. Kronfeld,[7] p. 54.
13. Abraham,[1] p. 899.
14. Kronfeld,[7] p. 73.
15. Magitot, A.: Ann. d'ocul. **168**:801 (Oct.) 1931.

But beyond that, I am actuated by scientific zeal and profound interest in running down the truth of the clinical phenomenon that I was the first to describe.

As stated clearly in the discussion of my paper read before the New York Academy of Medicine,[10] I find the high degrees of reactive hypertonia only in eyes that belong in group 2, which are "most, but not all, of the eyes affected with simple glaucoma." My material is not large enough to allow any final conclusions. Nevertheless, I should like to cite here briefly all the anterior chamber punctures performed at the University of Chicago Eye Clinic on eyes affected with glaucoma simplex which had normal or high normal tension at the time of the puncture. To eliminate the possible influence of my theory on the measurements made, I have not included the anterior chamber punctures done outside of Billings Hospital or those of which I could find no record in the "official" charts of the patients.

At the convention of the American Ophthalmological Society in 1930, I mentioned my results with the anterior chamber puncture in cases of simple glaucoma. When I read the proofs, I apparently overlooked one gross error.[16] I did not mean to say that I had done two hundred punctures in cases of glaucoma. What I meant was that I had done altogether approximately two hundred withdrawals of the aqueous in human eyes, and did not observe any untoward results. Of many of those I have no accurate records of tension, afterward. The number of glaucomatous eyes on which this test has been performed is, so far as I know, between fifty and seventy.

Table 3 shows that out of fifteen punctures performed *lege artis,* without previous medication, on eyes affected with simple glaucoma, twelve eyes (80 per cent) behaved as described in my previous papers; namely, the tension, which either was below 32 (six eyes) or exceeded this limit by 5 mm. or less (five eyes) or by 9 mm. (one eye) before the puncture, dropped first to 0 and then went up to values above 49 as the highest point in from seventy to one hundred and thirty-three minutes (average, ninety-seven minutes). The reactive hypertonia in these eyes was decidedly more pronounced than that in the control eyes of people of the same age. The higher degree of reactive hypertension can therefore be said to be characteristic of the large majority of eyes affected with simple glaucoma. It is, however, not found in every case of glaucoma simplex, as cases 9, 10 and 11 (table 3) indicate. Case 9, showing a highly myopic eye with a volume of the anterior chamber of 0.4 cc., may be just an apparent exception. I have observed no control eye with the same depth of anterior chamber, and therefore do not know how a nonglaucomatous eye of the same structure as that in

16. Kronfeld, P. C.: Tr. Am. Ophth. Soc., 1930, p. 151.

TABLE 3.—*Results of Anterior Chamber Puncture in Glaucomatous Eyes*

Case, Patient, Age, Sex	Tensions	Optic Nerves	Visual Fields	Tension (Anterior Chamber Puncture)			Volume of Fluid Obtained, Technic	Final Disposition
				Immediately Before	Immediately After	Highest After		
1 L. W. 72 Female	April, 1928: In clinic, tension about 50 in both eyes; not lowered by miotics. April 15: Admitted to hospital. Tension 22-27	L.E.: Total complete excavation	L.E.: Only temporal island left	L.E. 35	Not taken	56 (90 min.)	Volume not measured; technic apparently good	April 20, 1928: left Elliot trephine
	April 18: Anterior chamber puncture on left eye. Right tension remained below 24 even after dilation of right pupil (homatropine) to 5 mm. April 30: R.E. 30, in clinic. May 17: Readmitted to hospital. Right tension varied between 22 and 32; was highest about noon	R.E.: Total complete excavation	R.E.: Very extensive defects in central area	R.E. 34	Not taken	56 (100 min.)	Volume not measured; technic apparently good	June 5, 1928: right Elliot trephine
	June 1: Anterior chamber puncture on right eye							
2 A. N. 80 Male	June 15, 1928: In clinic, tension 28 in both eyes; under pilocarpine, 22-26. June 26: Tension of left eye not raised by dilation of pupil up to 6 mm. June 27: Admitted to hospital. R. = 15-19; L. = 19-28. June 30: Anterior chamber puncture on left eye. Discharged from hospital	L.E.: Total complete excavation	L.E.: Only temporal island left	L.E. 37	Not taken	71 (83 min.)	Volume not measured; technic apparently good	
	July 6: Anterior chamber puncture on right eye in clinic. Tension remained normal for some time and could not be increased by mydriatics. Later, anterior chamber became shallower. Tension rose again (35-45); lowered by miotics and strongly increased by mydriatics	R.E.: Total complete excavation	R.E.: Only temporal island left	R.E. 19	Not taken	56 (70 min.)	Volume not measured; technic apparently good	
3 J. McD. 65 Male	Feb. 14, 1929: Tension 33 in both eyes. February 15: R. = 27; L. = 33 in clinic. February 18: Admitted to hospital. Right tension varied from 22 to 36; left tension usually a little lower; but also rose to 36	L.E.: Total complete excavation	L.E.: Upper nasal step	L.E. Not done			.....	March 5, 1929: left Elliot trephine
		R.E.: Never seen	R.E.: Only temporal island left	R.E. 21	0	52 (100 min.)	Volume not measured; technic apparently good	Feb. 26, 1929: right Elliot trephine
	February 24: Anterior chamber puncture on right eye							
4 J. F. 75 Male	June 12, 1929: In clinic, tension 31-33 in both eyes. June 14: Admitted to hospital. R. = 27-31; L. = 23-27	R.E.: Shallow excavation; bending of the vessels; glaucomatous halo	R.E.: Paracentral arcuate scotoma	R.E. 31	Not taken	61 (95 min.)	Volume not measured; technic apparently good	June 25, 1929: right Elliot trephine
	June 15: Anterior chamber puncture on right eye							
		L.E.: Same as right	L.E.: Same as right	L.E. Not done			.....	June 18, 1929: left Elliot trephine

5 L. T. 69 Female	April 26, 1930: R.= 23; L. = 66. Pilocarpine ordered for L.E. April 28: R. = 22; L. = 27. April 29: R. = 25; L. = 22. May 6: R. = 23; L. = 36. Admitted to hospital. R. = 18-23; L. = 27-56 May 7: Anterior chamber puncture on right eye. June 3: R. = 20; L. = 8. June 17: R. = 26; L. = 10. Tension of each eye has been low at the few examinations since Oct. 14, 1932	L.E.: Total almost complete excavation	L.E.: Very extensive		L.E. Not done		.....	May 10, 1930: left Elliot trephine
		R.E., April, 1930: Normal physiologic excavation. October, 1932: Vessels displaced nasally; excavation reaches margin of disk on temporal side above and below	R.E.: Slightly constricted for red; extensive paracentral defects found by Bjerrum technic	R.E. 17	Not taken	52 (100 min.)	0.2 cc.; technic apparently good	
6 J. P. W. 56 Male	Jan. 4, 1930: Under homatropine, R. = 43; L. = 46. January 8: R. and L. = 27. January 16: R. = 42; L. = 45. Pilocarpine 2 per cent ordered four times a day. January 28: Has been without miotic for 1½ days; R. and L. = 33. March 1: Without miotic, R. = 31; L. = 29. March 3: Without miotic, R = 33; L. = 31. Tension without miotics remained around 30 (during morning hours); raised by mydriatics and definitely lowered by miotics. May 1: Admitted to hospital. Without miotic, R. = 26-33; L. = 23-31	L.E.: Normal disk	L.E.: Constricted for red		L.E. Not done		.....	Remains under miotic
	May 2: Anterior chamber puncture on right eye. Physostigmine started; tension remained below 23 in each eye. May 4: No miotic for 14 hours. R. and L. = 33	R.E.: Normal disk	R.E.: Constricted for red, fields unchanged	R.E. 31	0	57 (73 min.)	0.21 cc.; technic good	Remains under miotic
7 E. W. S. 71 Female	Feb. 6, 1930: Under homatropine, R. = 45; L. = 31. February 7: R. = 31; L. = 27. February 11: R. = 33; L. = 30. Right tension remained around 30 despite miotics (pupil, 1 mm.). February 24: Admitted to hospital. R. = 30-33; L. = 20-25	L.E.: Normal (Dec. 10, 1931)	L.E.: Normal		L.E. Not done			
	February 26: Anterior chamber puncture on right eye. Tension of both eyes remained below 29 for a year and then usually stayed around 30 in each eye	R.E.: Ampulliform excavation, reaching margin above, temporally and almost below	R.E.: Upper nasal step, almost superior hemianopia	R.E. 33	0	53 (105 min.)	0.12 cc.; technic good	Feb. 27, 1928: right Elliot trephine

TABLE 3.—*Results of Anterior Chamber Puncture in Glaucomatous Eyes—Continued*

Case, Patient, Age, Sex	Tensions	Optic Nerves	Visual Fields	Tension (Anterior Chamber Puncture)			Volume of Fluid Obtained, Technic	Final Disposition
				Immediately Before	Immediately After	Highest After		
8 L. F. 55 Male	July 3, 1929: R. and L. = 45. Tension varied between 36 and 45; not lowered by miotics	R.E.: Total complete excavation	R.E.: Severe degree of constriction	R.E. 36	Not taken	52 (80 min.)	Volume not measured; technic apparently good	Feb. 13, 1930: right Elliot trephine
	Feb. 10, 1930: Admitted to hospital; anterior chamber puncture on right eye	L.E.: Same	L.E.: Same		L.E. Not done		.....	March 8, 1930: left Elliot trephine
9 J. D. 45 Male	Dec. 2, 1930: R. = 36; L. = 66. Admitted to hospital. R. = 20-29; L. = 26-39	R.E.: Normal	R.E.: Normal	R.E. 29	0	45 (210 min.)	0.44 cc.; technic apparently good (except for fistulization)	Under miotics
	December 5: Anterior chamber puncture on right eye. Right tension remained 0 for 24 hours (fistulization), then rose again and varied between 23 and 41; left tension between 23 and 45	L.E.: Total complete excavation	L.E.: Temporal island left		L.E. Not done		.....	Under miotics
10 G. M. B. 58 Female	August, 1930, to February, 1931 (under Dr. B. Wilson's care): Tension consistently 45 to 60, not lowered by miotics, finally subsiding. March 30, 1931: R. = 23; L. = 31. Admitted to hospital. R. = 20-27; L. = 20-27	R.E.: Central physiologic excavation	R.E.: Lower nasal step, almost inferior hemianopia	R.E. 27	0	42 (88 min.) 49 (133 min.)	0.29 cc. obtained; technic good	
	April 4: Anterior chamber puncture on right eye. Tensions on both eyes remained normal (except one tension of 33 in L.E.) Jan. 5, 1932 Second anterior chamber puncture on left eye, Oct. 10, 1932 R. = 18; L. = 36. Admitted to hospital. R. = 18-23; L. = 20-29. Under atropine, tension of right eye unchanged, L. = 31-35. Operation advised	L.E.: Definite deep excavation, reaching disk margin above, temporally and below; bending of all vessels	L.E.: Almost inferior hemianopia	L.E. 23	0	31 (108 min.)	0.28 cc.; technic apparently good	Oct. 26, 1932: left Elliot trephine

11 T. H. L. 79 Male	June 13, 1931: R. = 33; L. = 56. Tension promptly lowered by mioties. Admitted to hospital. R. = 20-29. Jan. 27, 1932: R. = 33. Admitted to hospital. R. = 25-35; raised by mydriatics to 45 February 4: Anterior chamber puncture on right eye	L.E.: Shallow excavation extending throughout entire width of disk	L.E.: Severely constricted for white and red		L.E. Not done		.....	June 23, 1931: left Elliot trephine
		R.E.: Vessels displaced nasally; questionable excavation	R.E.: Lower nasal step	R.E. 27	0	36 (170 min.)	0.2 cc.; technic apparently good	Feb. 8, 1932: right iridencleisis
12 M. v. H. 53 Female	R. = 20-30.....	R.E.: Vessels displaced nasally; excavation approaching disk margin above, nasally and below	R.E.: Arcuate paracentral scotoma	R.E. 29	6	39 (60 min.)	Volume not measured; incomplete withdrawal	
	L. = 50-80.....	L.E.: Not visible	L.E.: Blind		L.E.		.....	Enucleation
13 D. M. 67 Female	Feb. 23, 1932: R. = 25; L. = 42. Admitted to hospital. R. = 19-25; not definitely raised by homatropine. L. = 25-33 February 28: Anterior chamber puncture on left eye February 29: Anterior chamber puncture on right eye	L.E.: Total complete excavation	L.E.: Only temporal island left	L.E. 41	0	61 (111 min.)	0.18 cc.; technic good	Under miotic
		R.E.: No pathologic excavation	R.E.: Normal	R.E. 23	0	12 (95 min.)	0.15 cc.; fistulization	Under miotic
14 C. G. 75 Female	Oct. 14, 1930: R. = 41; L. = 56. Tension lowered by mioties. Admitted to hospital. Tension of both eyes = 31-35 (under mioties	L.E.: Total complete excavation	L.E.: Severely constricted	L.E. 33	0	45 (155 min.)	0.15 cc.; technic apparently good; patient under miotic (pilocarpine 2% every 2 hours)	Oct. 21, 1930: left Elliot trephine
	October 20: Anterior chamber puncture on left eye	R.E.: Excavation extends to upper and lower disk margin	R.E.: Upper nasal step		R.E.		.....	Oct. 28, 1930: right Elliot trephine

case 9 would behave. If its tension curve is compared with the control eye of greatest anterior chamber volume in my series, it can easily be seen that the reactive hypertonia is more marked in the glaucomatous eye. Case 11 represents a real exception to the general rule, as the eye had all the characteristics of simple glaucoma, but showed only the reactive phase of a normal eye after the puncture. This case apparently represents one of the rare instances of retention glaucoma among the group of glaucoma simplex, according to my classification published in 1930. I am not in a position to say at this time in which group the left eye in case 11 belongs.

The possible value of the "anterior chamber puncture test" in making the diagnosis of primary simple glaucoma within a few hours is borne out by the cases presented. When opacities of the media prevent accurate ophthalmoscopy and perimetry and when the circumstances of the particular case make hospitalization and observation for a longer period impossible, anterior chamber puncture will often furnish valuable information. In case 5 it strongly suggested that the patient's right eye also had glaucoma, which was confirmed later by the formation of a pathologic excavation along with the development of characteristic field defects.

The diagnostic value of the test would be greater if the glaucomatous patient—because of his well known inclination to react with an increased tension to minor psychic shocks—did not frequently show a higher tension under the influence of the preparations for the anterior chamber puncture as in cases 1 and 2.

SUMMARY

The essence of my paper in 1930 [7] was:

> It seems, at least, very probable that the reactive hypertonia after anterior chamber puncture [beyond that of the nonglaucomatous eye] is a constant and essential symptom of eyes affected with simple glaucoma, a symptom even more characteristic than the intra-ocular tension. . . . More extensive investigations on a large material are necessary before we shall be able to make the diagnosis of the presence or absence of glaucoma from the result of the anterior chamber puncture.

I believe that the cases presented in this paper show that these statements were correct and justified. An estimation of the diagnostic value of the anterior chamber puncture by cool scientific observation and deduction rather than by bitter polemics leads to the conclusion that the anterior chamber puncture is a valuable provocative test which deserves a place among the various other procedures devised to diagnose early or latent primary simple glaucoma.

Clinical Notes

A STEREOSCOPIC FIXATION ATTACHMENT FOR THE PERIMETER

DONALD J. LYLE, M.D., CINCINNATI

Not infrequently the ophthalmologist desires to make an accurate study of the peripheral fields of an eye which has lost central vision. Charting of the central and paracentral fields is accurately done with stereoscopic instruments. Without binocular fixation field measurements are made with great difficulty and questionable precision when the patient cannot fix the eye under test.

Several devices have been developed for use in fixing the eye with a central scotoma, but they are, to my knowledge, hard to adjust, and the results are inaccurate. The old time method of placing the end of the index finger of the right hand (if the patient is right-handed) on the point of fixation and then instructing the patient to direct the eye to that point is still in use. This simple method, which seems to me to be as accurate as any of the mechanical devices and attachments, is still open to error, as the eye, denied central vision, constantly shifts and wanders.

The constant problem arising from the unsatisfactory results obtained from attempts at accurate charting of the peripheral fields in persons having no fixing power owing to central blindness led me to experiment on a more precise and time-saving means of recording these fields in this class of patients.

A simple attachment was devised for the Ferree-Rand perimeter with the principle of stereoscopic fixation. The black eye shield is removed from the head rest of the perimeter and a shield, which is attached to the end of a metal tube, is adjusted in its place (fig. 1). This shield is shaped so as to fit snugly over either eye, occluding all vision from that eye, save through the tube which leads to the fixing spotter. This spotter, which is part of the standard equipment, is illuminated from the usual source of light. The tube through which the fixing eye sights is hinged to the bottom of the spotter so that it may be moved in a horizontal direction for adjustment of pupillary distance for either eye.

The patient is placed at the instrument and requested to look into the fixation hole in the center of the arc with both eyes uncovered, so that the fellow eye can hold the centrally blind eye steady until the proper alinement is made. The attachment described is then adjusted over the centrally seeing eye, and the peripheral fields of the affected eye are charted as is customary.

As examples of the use of this sterescopic fixing attachment, two cases, one with an extra-ocular and the other with an intra-ocular lesion, are briefly presented.

REPORT OF CASES

A man, aged 38, was referred to me on Sept. 29, 1931, with the complaint of rapidly failing vision in the right eye. Examination showed vision of 20/200 in the right eye and 20/40 in the left. The media and fundi were normal in appearance. Refraction showed slight hyperopia of equal amounts in both eyes. The vision of the right eye, however, could not be improved by glasses. The left eye was corrected to normal. Extrinsic muscle balance was good. The pupils were equal and reacted

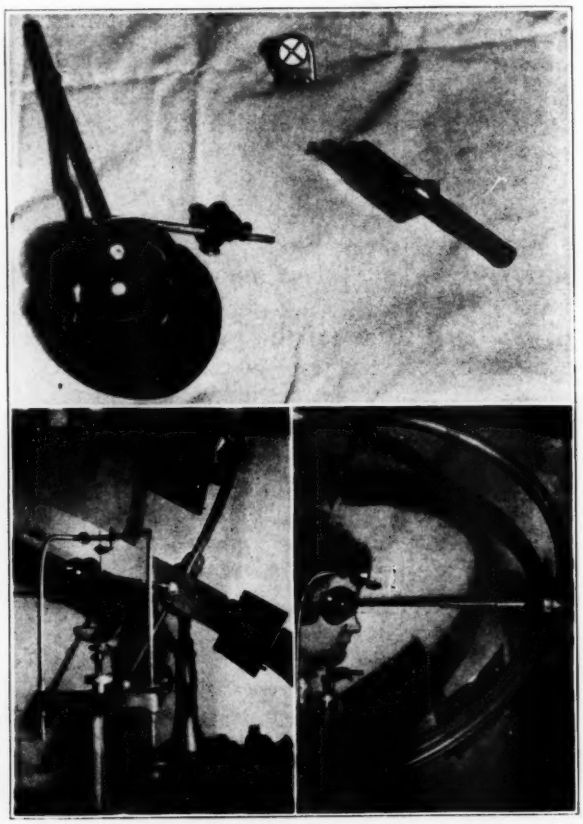

Fig. 1.—Photographs showing the fixation attachment and its use.

normally to all reflexes. Studies of the visual fields showed advanced bitemporal hemianopia. The patient was referred to a neurosurgeon, who found a large tumor in the pituitary gland. It was impossible to remove the mass entirely; so roentgen therapy was instituted with the request by the surgeon that visual fields be taken monthly in order to determine the progress of the case.

It was found, when the visual fields were taken for the first time after operation, that the macular area of the right eye was included in the anopsic area. Because of the urgency for a series of accurate determinations of the fields in order to determine the effect of roentgen therapy and the possibility of further surgical procedure, it was necessary to

devise some method of charting visual fields with the perimeter, or at least of accurate study of the peripheral fields, in an eye without central vision. Figure 2 shows the fundus and its peripheral visual field taken with the fixation attachment.

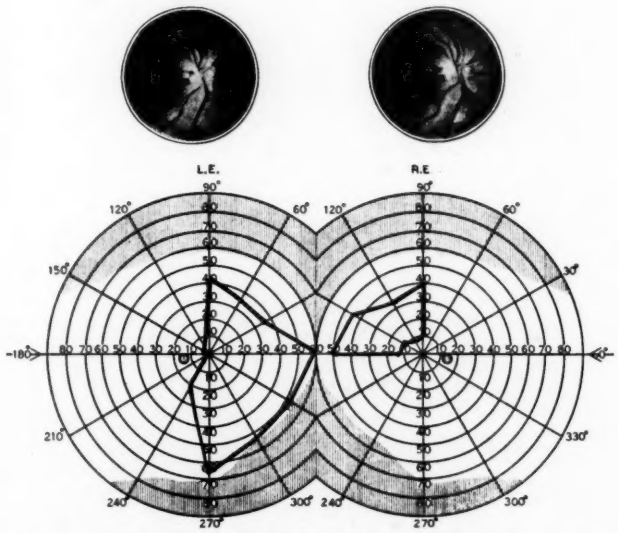

Fig. 2.—The fundus and its peripheral visual field taken with the fixation attachment.

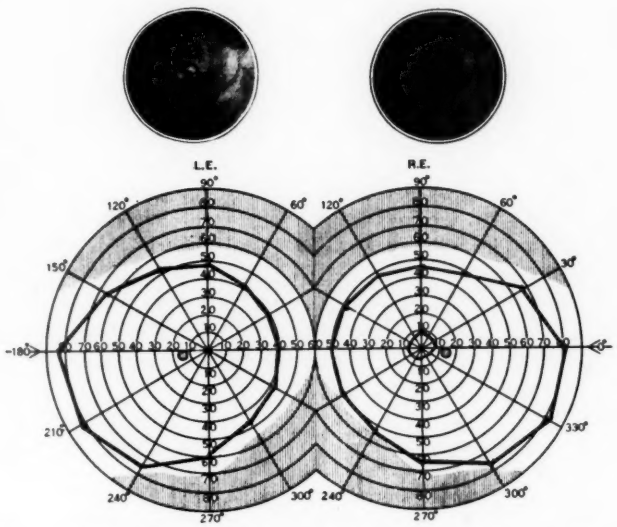

Fig. 3.—The retina and its peripheral field taken with the fixation attachment.

Another case in which central vision was absent and an accurate record of the peripheral fields was needed and made with the fixation attachment was that of a girl, aged 10 years, referred on Oct. 16, 1931, with the complaint of blindness of long standing in the right eye, occasional violent dizziness, severe headaches and astereognosis. An

intracranial lesion in the left parietal region was suspected. Examination of the eyes showed a small central patch of chorioretinitis in the right eye. Further ophthalmologic examination and other tests of the eyes gave negative results. Central and paracentral visual fields of both eyes and the peripheral fields of the left eye were examined. Although complete homonymous hemianopia was ruled out because of the normal records of the right paracentral field and the complete left field, the left nasal field was questionably constricted, and it was desired to determine the extent of the right temporal field as the case was further complicated by the fact that the child was left-handed, and therefore the aphasias, alexias, etc., usually found in the left parietal region in right-handed persons would not be present in a similar lesion in this case. Figure 3 shows a retinal photograph and a chart of the peripheral field taken with the fixation attachment.

ACUTE PURULENT ENDOGENOUS ENDOPHTHALMITIS

Report of a Case in the Absence of an Acute Systemic Infection

DAVID S. ASBILL, M.D., STATESVILLE, N. C.

Metastatic panophthalmitis is fairly common and generally follows some acute systemic infection, such as puerperal sepsis, cerebrospinal meningitis, pneumonia and measles. In this paper I am not concerned with panophthalmitis from such causes. I am considering only those rare cases of acute purulent endogenous endophthalmitis occurring in the absence of an acute systemic infection.

Selenkowsky and Woizechowsky [1] found that in rabbits and cats with many bacteria in the circulating blood the organisms invaded the vitreous of normal eyes. When the bacteremia was slight, however, trauma to the eye was necessary to bring about infection.

Oreste [2] reported a case of metastatic choroiditis the probable cause of which was an unknown lesion in the gastro-intestinal tract or kidneys. Betti [3] reported two cases following contusion of the globe, but the original focus was not found. Verhoeff [4] reported a case of metastatic intra-ocular mycosis. Terrien [5] reported a case of suppurating iridocyclitis with the gallbladder named as the focus of infection and the pneumococcus as the causative organism. Levine [6] reported a case of

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From the Davis Hospital.

Read before the Medical Society of the State of North Carolina, Eye, Ear, Nose and Throat Section, Winston-Salem, N. C., April 19, 1932.

1. Selenkowsky, J., and Woizechowsky, N.: Experimental Work on Metastatic Panophthalmitis, Arch. f. Augenh. **47**:299, 1903.

2. Oreste, A.: Metastatic Choroiditis, Ann. d'ocul. **159**:460, 1921.

3. Betti, L.: Colon Bacillus Infection of the Eye, Riforma med. **35**:784, 1919.

4. Verhoeff, F. H.: Metastatic Intra-Ocular Mycosis, Arch. Ophth. **55**:225 (May) 1926.

5. Terrien, F.: Metastatic Ophthalmia, Ann. d'ocul. **159**:460, 1922.

6. Levine, J.: Metastatic Bacillus Coli Panophthalmitis from Calculus Pyonephrosis, Arch. Ophth. **3**:410 (April) 1930.

metastatic Bacillus coli panophthalmitis from calculous pyonephrosis. Levine also mentioned a case of Dr. Goldstein's of New York in which B. coli ethmoiditis was the focus of infection. Van Fleet [7] reported a case of panophthalmitis due to a tonsillar infection.

The intensity of the infection varies. Sometimes the panophthalmitis rapidly perforates the globe; sometimes a hypopyon iridocyclitis produces eventual atrophy, and, again, an insidious but very serious form exists which in children often simulates glioma.

REPORT OF CASE

History.—G. M., a white boy, aged 6 years, entered my office on Aug. 30, 1930, with the chief complaint of pain and loss of vision in the right eye. The family history was irrelevant. The patient had had chickenpox, pertussis, grip, tonsillitis, influenza and occasional colds. These diseases never assumed serious proportions. He had not been ill since an attack of tonsillitis about two months before. There had never been any history indicative of disease of the paranasal accessory sinuses or gastro-intestinal disease.

The patient's eyes had never been subject to inflammation. His family denied that any injury had occurred to the affected eye. The child was very intelligent. He stated that he had never sustained any injury to his eyes.

Present Illness.—On Aug. 29, 1930, about 5 p. m., the patient complained to his parents that something was in his right eye. At that time the eye smarted and felt irritated. The father quoted the child to the effect that during the evening the electric lights had a "rainbow around them." The boy ate a small evening meal and went to bed. He slept until about 2 a. m., August 30, when he began to fret and cry with pain in the right eye. There was no discharge from the eye. The lids were not agglutinated or swollen. The patient slept no more, and the family physician, Dr. J. S. Talley, was called. The latter sent the child to me. The patient arrived at about 8 a. m., at which time he hung his head and held the right hand to his right eye. His expression was pinched and worn, and he was crying a little.

Examination.—The right eye showed swelling of both lids, especially the upper, with some engorgement of the veins of that lid. The palpebral conjunctiva was red, and the vessels were injected. No exudate was visible in the conjunctival sac. The ocular conjunctiva was slightly edematous, and the blood vessels were injected both circumcorneally and posteriorly. There was marked episcleral injection. The cornea was uniformly steamy and quite lusterless. Through the cloudy cornea were visible various-sized, cream-colored, posterior corneal deposits, but the latter could be made out only with difficulty because of the great cloudiness of the cornea. The deposits were present over the entire posterior corneal surface and were thicker in the inferior quadrant, though no hypopyon was present. The aqueous appeared turbid. The iris could be seen very indistinctly, and none of its finer structural details were discernible. The color of the iris was a hazy, muddy, yellowish gray. It could not be seen whether or not vessels were present on the iris. The pupil on the right side was 5 mm. in diameter, being twice the size of the left pupil. The left pupil reacted to light, in accommodation and in the consensual reflex. The right pupil did not react at all. Both eyes could be moved freely in every direction. The right eye was very tender to palpation, and the tension was obviously greatly increased. The cornea and conjunctiva showed no staining

7. Van Fleet, J. Flandreau: Endogenous Panophthalmitis Accompanying Tonsillitis, Arch. Ophth. **6**:426 (Sept.) 1931.

with a 2 per cent alkaline solution of fluorescein. There was no evidence of trauma. The lacrimal sac and puncta were normal. Vision of the right eye amounted to perception of movements at 2 feet (1.2 meters). The left eye and its appendages appeared normal with the usual methods of examination. Vision of the left eye was 20/20.

Ophthalmoscopic examination of the right eye was impossible by direct and indirect methods. Tension in the right eye was 40 mm. of mercury (Schiötz); in the left eye, 18 mm.

Transillumination of the right eye gave a uniformly diminished intensity of reflex as the light was moved around the globe.

The ears and nose were normal. No pus was seen in the right or left nasal fossa. The tonsils were slightly enlarged. A narrow, red, sharply defined area of injection was present on the anterior pillars, and yellow caseous material and pus could be expressed from the tonsils. Adenoids were present in the nasopharynx. Otherwise, the nose and throat were normal. Roentgenograms of the paranasal accessory sinuses showed a normal condition.

The general physical examination by Dr. C. W. Ashburn gave negative results except that on the skin of the legs there were several small furuncles. The latter were incised. Smears and cultures were made, and the laboratory report stated that many polymorphonuclear leukocytes and Staphylococcus aureus were present.

The temperature on admission was 99 F., the pulse rate, 90, and the respiratory rate, 20. A general physical examination otherwise gave negative results, as did a fluoroscopic examination of the chest.

Results of other laboratory examinations were as follows: Urinalysis gave negative results except for albumin, acetone, indican, hyaline casts and squamous epithelium; the reaction for each was one plus.

Examination of the blood showed: hemoglobin, 75 per cent (Dare); red blood cells, 4,500,000, and white blood cells, 11,500. The differential count revealed: polymorphonuclears, 87 per cent; small lymphocytes, 9 per cent, and large lymphocytes, 4 per cent. There were no transitional cells, eosinophils, basophils or abnormal cells present. Achromia was one plus.

The feces were normal. The Wassermann test of the blood, the intracutaneous tuberculin test and blood cultures gave negative results. The sedimentation rate was one hour (the normal rate is five hours to the 18 mm. mark).

Treatment consisted in hospitalization and the following measures: a laxative on admission, 5 cc. of sterile milk intramuscularly daily, soap suds enemas when necessary, hot water compresses to the right eye, atropine sulphate solution, 1 per cent, in the right eye after the hot compresses, acetylsalicylic acid, 5 grains every two hours, forced fluids by mouth, and a soft and liquid diet.

Course.—On August 30, the temperature was 102.6 F., the pulse rate 125 per minute and the respiratory rate 35 per minute at 4 p. m. The patient slept at intervals, but was restless for the greater part of the night. There was very little change in the appearance of the eye except that the inflammatory signs were slightly accentuated. Tension was 42 mm. (Schiötz) in the right eye and 18 mm. in the left.

On August 31, the temperature was 102.8 F., the pulse rate 130 and the respiratory rate 34 at 4 p. m. There were more swelling of the lids and more chemosis of the conjunctiva. There was fixation of the right eyeball with moderate proptosis. A small hypopyon was present in the right eye, and all the inflammatory signs were increased. Tension was 60 mm. (Schiötz) in the right eye and 20 mm. in the left.

On September 1, the temperature was 103 F., the pulse rate 132 and the respiratory rate 20. The patient was more restless and complained at intervals of pain in the right eye. He held his hand to the right eye most of the time. The eye was more tender to the touch, and the tension was still more elevated to palpation. Marked proptosis of the right eye was present. Enucleation was advised, but the patient's family refused. They also refused incision of the cornea for drainage.

On September 2, the patient spent a very poor night, and suffered at intervals with pain in the right eye. A mass of yellow exudate filled the anterior chamber. The eye was of stonelike hardness to palpation. All the inflammatory signs were greatly increased. By 10 a. m. a small perforation occurred near the periphery of the cornea with the liberation of cream-colored pus. Under nitrous oxide and oxygen anesthesia and sterile precautions, a free incision was made in the cornea, and a large amount of pus escaped through the wound. A smear and culture were made from the pus. The laboratory report was that the hemolytic streptococcus was found. Sterile dressings were applied.

Condition of the eye on Sept. 2, 1931, following perforation of the cornea.

On September 3, the patient was far more comfortable. Urinalysis gave negative results. Profuse drainage occurred from the right eye, which had to be dressed several times daily.

The patient made a rapid convalescence and was discharged from the hospital on September 5 with a normal temperature. His eye was dressed as required. Drainage ceased in about a month. On November 22, a tonsillectomy and adenoidectomy, together with enucleation of the right eye, were performed under ether anesthesia. Recovery from the operation was normal and uneventful. The patient began to wear an artificial eye on Jan. 1, 1931. Cultures were made from the interior of the removed tonsils, and hemolytic streptococci, staphylococci and pneumococci were found. Sections of the removed phthisical eye showed only sclera with a mass of scattered pigment and exudate adjacent.

The source of the infection could not be found with absolute certainty. However, as the sinuses, teeth and all other possible sources of infection seemed to be ruled out, it was decided that the chronically diseased tonsils were probably the responsible foci of infection.

DISCUSSION

Dr. C. W. Banner, Greensboro, N. C.: This is one of the cases that one frequently finds in practice in which one cannot locate the cause. I think that in this particular case there were two causes that might be considered. One is apparently offset by the analysis of the blood. There was a history of abscesses on the legs, and yet there was no infection of the blood stream. It may have been overlooked by the laboratory technician, but probably it did not show up.

The other possible source of infection was the tonsils, as Dr. Asbill has suggested. This was one of those cases in which one does not know just what to do because of opposition from the family. Dr. Asbill was perfectly justified, in my opinion, in wanting to remove the eye at an early stage of the disease, but with the opposition of the family of course he could not.

I have removed the eye in quite a number of cases of panophthalmitis. There is some question whether it is better to remove the eye in the early stage of the disease or wait, as in this case, until the infection has run its course. I have never observed more than the usual reaction following removal of the eye in these cases.

If one is careful not to rupture the eyeball, one will not have much difficulty. Sometimes when there is an ulcer and the eyeball ruptures an infection occurs and the danger is increased. I try to be very careful in my dissection, and apply no pressure to the eyeball that might cause it to rupture.

I had an instrument made for cutting the nerve just back of the eye. It is a small modified Lewis tonsil snare with just sufficient curve to pass behind the eye without pressure or traction. It closes the vessels, and very little hemorrhage occurs. I have been using it for three years, with a great deal of satisfaction.

There are two things in the treatment of the patient that I am inclined to criticize, although I do not think that any treatment would have saved the eye. In the first place, in an eye that has a dilated pupil with increased tension, a hazy cornea and vision of rainbow circles around the light, as reported in this case, I should hesitate to use atropine. I do not think that it did any harm, but am a little afraid of it.

Another thing that interested me was the use of hot applications in a case in which a pus infection was present. I think that with this treatment one provides an incubator for growing germs. I should be more inclined to use cold applications in the beginning of a case of this kind.

Dr. V. K. Hart, Charlotte, N. C.: Dr. Banner, would you mind passing your instrument around? I imagine that some of the men want to see it.

Dr. V. M. Hicks, Raleigh, N. C.: The hemolytic streptococcus is a queer "varmint," and one may expect it to do anything. Dr. Asbill established the fact that the eye was lost from infection with the hemolytic streptococcus, the origin of which was unknown. The infection that existed in the tonsils at the time of the first examination, it seems to me, was prima facie evidence that the tonsils should be removed.

I am inclined to believe that nose and throat specialists have been somewhat browbeaten in the discussion of radical operations on the head in a serious condition such as this.

Not infrequently I deal with an ocular condition that I feel requires a radical procedure, and I can get a nose and throat specialist to say with a good deal of hesitation only that he thinks that it should be used.

The general physicians have kept at the issue of whether a tonsil is normal until the nose and throat specialists are raising the question themselves. Experience should be the best teacher, and when one is dealing with an infection

that is intra-ocular, I think that it is time enough to forego conservative methods; surely the removal of tonsils does not prevent the possibility of giving the patient the full benefit of any other treatment that might be instituted.

I think that is particularly true in cases in which there is a question of whether or not a sinus is blocked or infected and whether or not it is getting sufficient drainage. It is commonly true that a nose and throat specialist refuses to open a sinus. I am sure that I have seen many cases of infection miraculously cured by the simple opening of an antrum that did not appear infected to the nose and throat specialist.

I therefore plead in such cases as this that heroic methods should be instituted and every vestige of the possible cause removed. I think that frequently one loses the opportunity to obtain a permanent beneficial result by failing to use heroic methods.

DR. DAVID S. ASBILL: The snare which Dr. Banner presents is interesting, and I think that I shall get one.

Atropine would be absolutely contraindicated in primary glaucoma. However, in the present case I felt that the condition was undoubtedly a secondary glaucoma, as I believe that primary acute congestive glaucoma is unknown below the age of 10 or 12 years. As the glaucoma was evidently due to the iridocyclitis, the treatment of the glaucoma was the treatment of the iridocyclitis, which in this case was the use of atropine. Had the condition of the eye warranted, repeated paracentesis of the cornea would have been employed to control the tension.

These are the reasons why atropine was used and my line of reasoning in instituting this type of treatment. The hot compresses were used for their soothing effect, as cold compresses were not tolerated.

I think that Dr. Hicks made a good point in stressing the importance of doing what one is going to do in time. My reaction at first was a wish to remove the patient's tonsils. However, the thought came to me that the disease was already so far advanced that probably nothing would benefit the child. If I removed the tonsils and the ocular infection nevertheless went on and the eye was lost, I would be throwing a certain amount of discredit on the operation of tonsillectomy, which already has had ample criticism.

CYST IN THE VITREOUS

Report of a Case

CHESTER E. HURWITZ, M.D., BALTIMORE

Cysts in the vitreous are very rare. I have been able to find only seven cases reported in the literature to date. However, it may be possible that cysts in the vitreous are not quite as uncommon as one thinks they are, as they may be easily overlooked. The patient may have no subjective symptoms whatever and may never get to the ophthalmologist, or he may come only for a refraction, and the refractionist

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Read before meeting of Section of Ophthalmology, Baltimore City Medical Society.

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

easily fail to see the cyst. In the present case the patient was seen and his refraction tested three different times by capable men, and each time the cyst was overlooked.

REPORT OF A CASE

A girl, aged 13 years, came to me on July 9, 1931, complaining that the glasses she had gotten two and one-half years before were no longer satisfactory. She had occasional frontal headaches, but no other symptoms. On cursory examination of the eyes nothing abnormal was seen. She was given a mydriatic and told to return for retinoscopic examination. On July 13, retinoscopy was done and the following correction given: right eye, —3.00 sph. ⌒ —2.00 cyl. axis 180°; left eye, —2.25 sph. ⌒ —2.25 cyl. axis 180°.

At this last visit, examination with the ophthalmoscope revealed the presence of a cyst floating freely in the vitreous. The cyst was round, about the size of the optic disk. The vitreous must have been quite fluid, for no matter in what position the patient held her head the cyst always sank to the lowermost level of the eye. When the patient lay face down, the cyst was seen almost against the lens; when she lay looking up, it was found far back in the vitreous. The cyst was not connected or attached to any structure in the eye. On motion of the eye it moved from place to place, but rather quickly returned to the lowermost part. The specific gravity was therefore somewhat greater than that of the vitreous. It rotated freely in all directions but always settled with the same side down, that side evidently being the heaviest. The cyst contained on its surface many irregular spots of pigment, most numerous on the under surface. The capsule, if there was any, was very thin. The contents of the cyst were transparent, the posterior wall being visible through the cyst, and the posterior pigment spots differentiated from the anterior by a parallax motion. The retinal blood vessels could also be clearly seen through the body of the cyst.

An attempt was made to examine the cyst with the slit-lamp, but it was impossible to get it in view as it always sank below the field. However, with the patient recumbent and a narrow beam of light thrown into the eye when the pupil was dilated to its maximum, the cyst was seen as a silver-white, glistening sphere, pigmented in its lower part, and with a shred or two of connective tissue running off loosely from its upper portion.

REVIEW OF LITERATURE

A case much like the foregoing was reported by Tansley,[1] in 1899, in a 17 year old boy. This seems to have been the first case ever reported. The cyst, somewhat less spherical than that in the case reported here, also floated freely in the vitreous, had a few pigment spots and lines on its surface, and sank to the bottom of the eye when the latter was quiet. In discussing this case, Alt thought that the floating body was a tumor of the ciliary process which had broken loose and that it resembled an adenoma undergoing degeneration. Randall said it might have been a cystic growth at the site of a coloboma, which was freed from its detachment and set loose in the vitreous by closure of the congenital cleft.

In 1903, Troncoso [2] reported a case in a 15 year old girl. The cyst was oval, was in the anterior part of the vitreous, was freely movable

1. Tansley, J. O.: Tr. Am. Ophth. Soc. **9**:507, 1899.
2. Troncoso, Uribe: Ann. d'ocul. **130**:341, 1903.

and gradually settled to the bottom of the eye when the latter was at rest. Because of the pigment spots on the wall of the cyst, it was thought to be derived from the uvea.

In 1913, Shine [3] presented a case before the Section of Ophthalmology of the New York Academy of Medicine. The patient came because of asthenopic symptoms which were later cleared up by the use of proper glasses. The cyst floated freely on movements of the eyeball, but on cessation of movements always returned to the same point just behind the ciliary body below or slightly to the temporal side. The cyst was globular, of steel gray color, and semitransparent. Shine thought it was congenital and composed of epithelial tissue, an outgrowth from the ciliary body which became detached. The fact that it always returned to the same position suggested a small invisible filament still attached to the ciliary body. Fridenberg recorded at this meeting a similar case in which the cyst was slightly pigmented and had slow motion, but no strand could be observed.

In the same year, Brewerton [4] reported a case in which, after treating a 49 year old man for a marked iridocyclitis, he found two cysts in the vitreous. These were oval, were quite transparent, but did not float. There was also a detachment of the retina in this case, and later the vitreous became so full of opacities that the details of the fundus could not be seen.

In 1929, Scarlett [5] presented a case before the American Ophthalmological Society. The patient, an 8 year old girl, complained of intermittent blurred vision for six months previous to being seen. The vision varied from 6/6 to 6/30. The cyst was irregularly oval, translucent and situated toward the anterior temporal quadrant. It was covered by numerous pinpoint black dots. When the eye moved quickly, the cyst assumed various positions, but righted itself into a constant vertical direction as soon as the eye resumed its normal position. It was not attached to any structure in the interior of the eye. The vitreous was apparently fluid. The disk and the rest of the fundus were normal. Because of the pigment spots, Scarlett diagnosed this a congenital uveal cyst.

At the same meeting Hill recorded another case in a middle-aged man who came to him because of poor vision. The cyst was globular, floated in the vitreous without any apparent attachment, and moved only slowly with the movements of the eye. The nerve head was slightly pale in 1925, and became atrophic by 1928. Hill did not say what he thought to be the origin of this cyst.

In the case reported here the first thought was that the cyst was a Cysticercus. On closer examination, however, it was found to be too homogeneous, and there was no movement present within the cyst. As the patient has now been under observation for eighteen months, and there has been no noticeable change in the condition all this time, the possibility of Cysticercus is precluded. My own feeling is that the cyst originated congenitally in a ciliary process and became detached into the vitreous.

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3. Shine: Arch. Ophth. **42**:398, 1913.
4. Brewerton: Tr. Ophth. Soc. U. Kingdom **33**:93, 1913.
5. Scarlett, E. P.: Tr. Am. Ophth. Soc. **27**:154, 1929.

A RETRACTOR FOR THE KRÖNLEIN OPERATION

W. H. Wilmer, M.D., and H. F. Pierce, Ph.D., Baltimore

The accompanying photographs illustrate the details of a retractor [1] which was designed especially for use in the Krönlein operation to retract the orbital contents medially. Its proportions are based on measurements taken from plaster casts of a number of normal adult

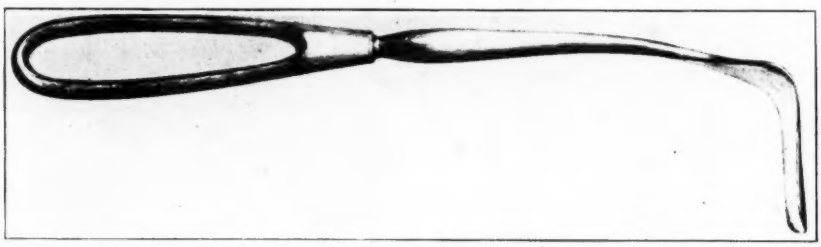

Fig. 1.—Retractor for the Krönlein operation.

Fig. 2.—Front of the blade.

orbits. Its advantages over the usual narrow retractor are: (1) The blade is broad enough to protect the eyeball completely while the lateral wall of the orbit is being sawn through; (2) the shaft is so curved that it will not touch the bridge of the nose when the contents of the orbit are drawn toward the medial wall, and (3) the blade is so proportioned that the outer edges of the tip touch the floor and roof of the orbit, thus preventing the center of the tip from bruising or lacerating the optic nerve.

From the Wilmer Institute of the Johns Hopkins University and Hospital.

1. This retractor is made by the Murray-Baumgartner Company, Baltimore.

Ophthalmologic Review

DIABETIC RETINITIS

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Diabetic retinitis is a term applied to an ophthalmoscopic appearance of the retina of a diabetic patient in whom there are exudates, hemorrhages or both. The term is a misnomer, since there is no actual inflammation and the findings are not pathognomonic of diabetes. Dabney (1926) expressed the belief that this condition is a distinct entity, while Wagener and Wilder agreed with the vast majority that it never occurs in uncomplicated cases. It was only four years after Helmholtz introduced the ophthalmoscope that Jaeger (1855) described the first case on record. Fourteen years later (1869) this same author described the condition again in a man only 22 years of age. In the plates accompanying the latter case, the author definitely differentiates diabetic retinitis from that associated with albuminuria.

It remained for Hirschberg, in 1890, to classify the various types as follows:

1. The characteristic inflammation of the central retina with small light spots and minute hemorrhages, "retinitis centralis punctata."

2. Hemorrhages into the retina with subsequent inflammatory changes and degeneration.

3. A rare form in which there was a pigmentary degeneration.

It will be noted that he considered the disease as an inflammation.

Much has been written since Hirschberg's paper, and many theories have been advanced and disproved as to the method of production of the condition and the treatment of the disease, but today there still are wide differences of opinion.

It is an accepted fact that the changes in the retinas of diabetic patients do not occur before the age of 40, except in a few rare cases (Cammidge, 1930; Cohen, 1929), and more women are included in the reports than men (Shepardson and Crawford). Heredity is found to be a great factor in diabetes mellitus (Cammidge, 1930). Wright

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Thesis submitted to the Faculty of Ophthalmology of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science (M. Sc. Med.) for graduate work in Ophthalmology.

reported several cases in which he traced the condition for several generations. In one family which he traced, diabetes had been present in three generations preceding the patient, and although none of the patient's children were diabetic, all were bleeders (six) and three had died of hemorrhage. There is no explanation offered as yet of the relation of the profound bleeding in these patients to the hemorrhages in the retinas of diabetic patients. The character may be dominant or recessive and is transmitted according to the law of Mendel. Cammidge (1928) stated that when the disease is transmitted as a dominant character the course is mild, but when it is transmitted as a recessive character the disease is more severe from the onset.

THE INSTANCE OF RETINAL DISEASE IN DIABETES

The percentage of diabetic patients having retinal disease varies with the author. Cammidge (1930) found only forty-eight cases in a thousand examined, and he quoted Joslin's cases as 5.2 per cent, von Noorden's as 17 per cent and E. Graefe's as 13 per cent. Most of these patients had suffered from the disease for at least three years (Shepardson and Crawford) before they showed any manifestations of retinal disease. Arteriosclerosis with (Onfray, 1919, and Spalding and Curtis) or without (Cohen) hypertension is the most common accompaniment of the condition and is usually considered the exciting factor in the production of the hemorrhagic retinitis in diabetes. Spalding and Curtis reported sixteen cases of retinitis and discussed only two with hypertension; however, examination of the table included in their article revealed eight cases with a diastolic pressure of 100 or more. Morrison and Bogan stated that the diabetes itself is an etiologic factor in the production of the vascular disease so often accompanying retinitis, and Mosenthal suggested that there is a possibility that a high blood sugar "in itself and indirectly" may exert an unfavorable influence on the development of the arteriosclerosis by virtue of a toxic action produced by "desiccation through polyuria."

The presence of kidney disease, which is not an uncommon condition in patients of the age under discussion, sometimes complicates the diagnosis. Some authors believe that all diabetic patients have renal deficiencies (Shepardson and Crawford). Cammidge (1930) stated definitely that nephritis has some bearing on the retinitis; however, he does not believe that it is sufficient merely to make a urinalysis, but that an efficiency test of the kidneys is imperative to determine the presence or absence of nephritis. The findings of some research workers (Shepardson and Crawford) indicate definitely a deficiency in renal function in diabetes. Holloway, however, in a discussion of H. Friedenwald's paper, quoted Foster Moore as saying that hemorrhages are not

dependent on an increased blood pressure such as would accompany renal inefficiency, but on the impaired nutrition of the vessel walls and on the changes in the blood.

The symptomatology present is entirely dependent on the location and extent of the lesion. Unless there has been a large hemorrhage or unless the macular region has been involved by larger or smaller lesions, the patient may not be aware of the existing disease until it is quite well advanced. The fundus picture is, of course, the outstanding feature of the disease, yielding, sometimes, the first knowledge of the condition, and it is often discovered quite by accident. Much has been written describing the fundus, and the descriptions vary decidedly, but there are some characteristics that are conspicuous and are generally accepted. The most distinctive features are the hemorrhages and exudates with an associated arteriosclerosis and sometimes an edema of the retina or a retinitis proliferans.

PATHOLOGY

Altnow listed the pathologic changes in the retina as follows:

1. The retina is redder than normal.
2. There are punctate hemorrhages in the retina.
3. There are grayish-white spots at the macula.
4. There may be a fan-shaped figure at the macula.
5. There may be an arteriosclerosis of the retinal vessels.
6. There may be a choroidal arteriosclerosis.

The hemorrhages vary in character, as is evidenced by the different descriptions in the literature. Some authors (Garrod) describe them as "guttate rather than flame-shaped." Gifford said that they occur along the larger vessels not far from the papilla. Cohen said that the hemorrhages are due to a diapedesis of the red corpuscles from the terminal capillaries in the deeper layers of the retina. This would account for the punctate type of hemorrhage so commonly seen.

It is quite universally accepted that arteriolosclerosis is an almost constant accompanying feature of diabetic retinitis, Cohen finding that the changes are present microscopically before they can be seen ophthalmoscopically. Cammidge (1930) quoted Borchardt as saying that the vascular changes are not different from an ordinary sclerosis.

The exudates may occur small and singly or in small, nonconfluent groups (Gifford). Heflebower described these exudates as brilliant and usually occurring in or about the macular region, but he said that they may be yellow and quite remote from the macula, and large and irregular in outline. This he believed is due to a coalescence of a number of small plaques. Onfray (1919) offered as an explanation

of the formation of these lesions the following: "the polycyclic plaques" are probably due to unabsorbed serous products altered by toxic agents in the blood. The brilliant white spots, isolated or conglomerated, are probably due to fatty deposits. R. Foster Moore (1920) depicted these exudates as having distinctive characteristics, being "sharply cut, often soapy, solid or waxy looking, with a tendency to form an irregular ring well wide of the macula." He said that there is seldom a star figure, and when it does occur, it lacks symmetry. Edema is not a common finding, but Altnow reported two cases. Cammidge (1930) declared that this is the result of the osmotic pressure change in the tissues because of the excess sugar in the blood. Pigmentation is not considered in most of the literature, but Foster Moore (1920) said that "circular retinal pigment is not rare late in this disease."

The pathologic changes in the retina associated with diabetes mellitus vary, but the most common lesions are hemorrhages and exudates in the retina and sclerotic changes in the vessel walls. The hemorrhages may be in any or all of the layers of the retina (Shepardson and Crawford). The exudates are diversely described. Shepardson and Crawford considered them "hyaline-like deposits" in the nerve fiber or nuclear layers, probably resulting from coagulated fibrin, and the larger plaques as probably due to the action of the toxins in the blood on unabsorbed serous products. The brilliant white lesions are undoubtedly due to fatty deposits (Onfray, 1919). J. S. Friedenwald wrote: "on section the white dots are found to be masses of colloid in the outer layers of the retina," but he does not think that they are different from the microscopic appearance of the exudates associated with albuminuric retinitis.

As regards the arteriosclerosis in the retinal vessels, Cohen said that the capillaries show local areas of thickening microscopically in cases in which the choroidal and retinal vessels appear ophthalmoscopically normal. He further stated that retinal arteriosclerosis is just a part of a generalized systemic arteriosclerosis. Some sections of the eyes which this author examined showed a definite thickening of the vessel walls in the choroid as well as in the retina and iris and an endarteritis obliterans of the long posterior ciliary artery.

As regards the retina itself, after a time there is a degeneration of the ganglion cell layer (Shepardson and Crawford). Preretinal hemorrhages are sometimes followed by retinitis proliferans (Gifford). Weisser expressed the belief that the pathologic changes in the retina occur late, and also that these changes are only part of an advanced generalized tissue change in the body.

Diabetic retinitis must be differentiated chiefly from the retinitis of simple arteriosclerosis and from that associated with albuminuria.

Since arteriosclerosis is a common accompaniment of diabetic retinitis, and since the vessel changes often occur in albuminuria and are the outstanding feature in arteriosclerotic retinitis, it is frequently difficult to make a definite diagnosis from the retinal picture alone. The extreme vascular changes are the most prominent part of the retinal picture in arteriosclerotic retinitis; the hemorrhages are relatively large, situated near fairly large vessels, and may extend into the vitreous. The patients are usually older and have an established hypertension. In albuminuric retinitis the retina appears to be affected with an acute condition; the hemorrhages are splashlike or flame-shaped; they are relatively large in a retina that is profoundly edematous; about the macula and papilla there are white exudates which have a soft edge, cotton-wool appearance (Chaufford, Grigart and Nida) and take on a star-shaped figure at the macula. This type has no respect for age; that is to say, it occurs in childhood as well as in adult life. In diabetic retinitis there are hemorrhages that are guttate rather than flame-shaped (Garrod); they occur throughout the retina and not always near a vessel. The exudates, which are sharply defined, soapy or waxy in appearance (Burch), are usually small unless they coalesce to form larger lesions, and then their edges are crenated, which fact supports the idea of their formation (by coalescence). The retina about these lesions is apparently normal (Weisser). There is seldom an orderly arrangement of the exudates, as there is in albuminuric retinitis, except that in rare cases there has been a fan formation (a partial star) at the macula (Altnow). Diabetic retinitis has rarely been seen before the age of 35. Since the ocular changes in these three conditions are more or less related by virtue of their vascular background and since all three may occur in the same patient, it is, as said, sometimes difficult to state positively which is the predominating factor in causing the retinal changes.

THE QUESTION OF TREATMENT

Most of the treatment of diabetic retinitis is directed toward the general diabetic condition. Cammidge (1930) expressed the belief that when the blood sugar is maintained within normal limits by the restriction of diet or the administration of insulin, or both, there is an improvement in the retinal condition. The effect of insulin in hemorrhagic retinitis is questionable. Carter, and also Davis, quoting Graefe, advised caution in its use, because they had noticed an accentuation of the hemorrhagic condition. A group of French writers (Chaufford, Grigart and Nida) observed immediate improvement in a case of central punctate retinitis which they accredited to the influence of insulin on the metabolism of fats. The white spots diminished in proportion to the reduction of fats in the blood. This group believes that insulin

acts not only on the fats in the blood, but also on that in the tissues. In a personal communication to Dabney (1930) concerning the treatment of "diabetic retinitis with angiosclerosis," de Schweinitz stated that iodides are an aid to clearing up the hemorrhages; that he preferred sodium salt in the absence of syphilis; that he would suggest calcium lactate internally to prevent, if possible, the recurrence of hemorrhages, and that insulin has been advantageous in some cases. Dabney (1930), however, found that insulin was no aid in controlling the hemorrhages. Cohen reported that in some cases there is a permanent pathologic condition in the retinal vessels which progresses during the use of insulin and the improvement of the patient's general condition. In considering the idea of endocrine dyscrasia, Cammidge (1930) has used parathyroid hormone, but as yet there is no report available on the results. He found a deficiency in the blood calcium, but the administration of calcium, in the presence of a renal disease, had no effect on the hemorrhagic condition of the retina. He found, too, that there was a rapid improvement of vision as the blood and urine findings became normal under treatment.

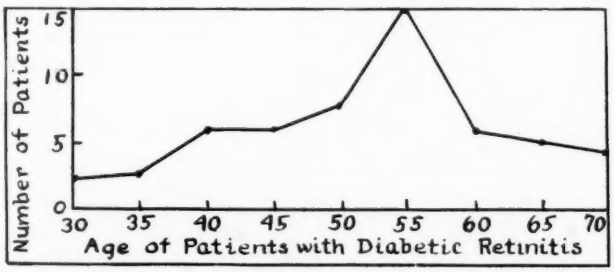
Recurrent preretinal hemorrhages lead to blindness (Dabney, 1930), and sometimes vitreous hemorrhages are followed by retinitis proliferans (Gifford). Other lesions influence vision by virtue of their position. In the macular region their effect is very destructive, while in the periphery they are not as pernicious. The prognosis as to life is not as definite as in albuminuric retinitis (Shepardson and Crawford), and many think that it is good (Dabney, 1926, and Onfray, quoted by Gradle). Gifford offered seven years as the expectancy of life after the occurrence of the retinal hemorrhages. Foster Moore (1920) quoted Nettleship as having forty-eight patients with diabetic retinitis, nineteen of whom died within two years after the discovery of the retinal hemorrhages, and eighteen within from two to eight years. Cohen stated that patients often die of apoplexy and hemiplegia as the result of vascular pathologic changes. He agreed with the vast majority that the retinal arteriosclerosis is only part of a generalized sclerosis, and that the appearance of the retinal vessels is an index to the condition of the vessels of the brain.

THE ANALYSIS OF SIXTY-EIGHT CASES OF DIABETIC RETINITIS

At the University Hospital of Western Reserve Medical College in Cleveland there were three hundred and ninety-eight diabetic patients under treatment. Most of them were dispensary patients and many were foreign, which deducts from the possibility of cooperation. I have seen sixty-eight patients because of ocular complications, and of this group, fifty-eight showed definite pathologic changes in the retina.

There were forty-three women and but fifteen men, the preponderance of women being due probably to the fact that women avail themselves of the opportunity for treatment more readily. The ages of the patients ranged from 31 to 73 years. A 36 year old patient had a well developed pulmonary tuberculosis, so that whether this prevented a purely diabetic retinitis might be questioned.

Thirty-six of the patients were being treated with insulin, while the remaining twenty-two were taught accurate methods of calculating their diets. The tension was taken (Schiötz) for each patient prior to dilatation with eucatropine (euphthalmine) for examination of the fundus. There was a wide variation in the tensions, from 6 to 35 mm. (one patient showed a tension of 105 mm., but this was due to a secondary glaucoma following an injury some years previously. The patients who were treated with insulin showed less variation, but,



Graph showing the increased tendency to pathologic changes in the retina between the ages of 50 and 60.

on the whole, their tensions were higher than those of the patients who were not taking insulin.

Renal function tests were not possible, but an albuminuria of varying degree was found in thirty-three patients. The patients of a hypersensitive type considered had a systolic pressure of 150 or over and a diastolic pressure of 100 or over. That is to say, a patient with a blood pressure of 140 systolic and 100 diastolic was considered to have a hypertension. Thirty-four patients were included in the hypertensive class, nineteen of whom were taking insulin and fifteen of whom were not.

The visual acuity varied from normal to light perception. It is remarkable to find normal vision in a patient whose fundus is literally "peppered" with hemorrhages and whose macular region is hemorrhagic, with only the brilliant reflex of the fovea to offer a definite landmark as to its location. It is probably this character of the disease, viz., the extensive pathologic changes with good vision, that is responsible for the discrepancies in reports as to the true duration of the retinal condition.

Many patients have an extensive hemorrhagic condition of the retina and are not aware that anything is wrong. The appearance of the fundi of the patients examined coincided with the descriptions of the authors mentioned; there was no orderly arrangement, and the lesions were found anywhere in the retina; they were superficial or deep, central or peripheral or a combination of any or all of these. When the eye was near emmetropia (when the ophthalmoscope allowed for focusing in values of 1 diopter), it was possible to see hemorrhages in the several layers of the retina. The hemorrhages were dotlike or striate. In three fundi there were flame-shaped hemorrhages which were of a darker color and denser than usual, owing to a large amount of blood; these patients had albumin in their urine. Two of the patients, both in the early forties, showed large preretinal hemorrhages, and each had only a "very faint trace" of albumin. Exudates were not always present. When observed, they were yellow or white and widespread in the retina, although the most common site was the papillomacular area. Pigment was not frequently seen, and when it did occur, it was fine in character and surrounded the areas of exudate. There were just as many exudative areas in the retinas of patients who were taking insulin as in those who were not, and no improvement was noted in the lesions during the ten months' study. Some of the patients who were taking insulin and who occasionally experienced some reaction or had a typical "insulin shock" had marked aggravation of the hemorrhagic condition in their eyes following such an attack. Many times there was little question but that there was improvement in the general diabetic condition manifested by the approach of the blood sugar to within normal limits and by the absence of glycosuria, and yet the ocular condition did not improve in like proportion regardless of whether the general improvement was accomplished by diet or insulin, or by both.

Practically all the retinas showed vascular pathologic changes, but some of the patients with extreme vascular sclerosis showed no hemorrhages, and in some, in whom the conditions were ophthalmoscopically mild, there were hemorrhages of various degrees. Ray said that the degenerative changes in the arteries "are the result of an excessive effort on the part of nature to repair the damage to the blood vessels by chronic toxemia," yet many of these older patients never showed an acetonuria, and children who became very toxic showed no changes in the fundus. (There were no cases of lipemia retinalis.) The idea, however, that a toxin produces the pathologic changes in the vessels seems logical, but the process whereby this is brought about demands some explanation. The toxemia is probably the result of some metabolic disturbance aside from that which occurs from the diabetes per se. Old foci of infection are often seen, and intestinal stases are very

common. These toxins may act as a stimulant to the endocrine system and disturb its sensitive equilibrium. Stimulation of the suprarenals, which respond so readily, results in vasoconstriction, and this is the first step in the production of hypertension, for with spasm of the capillaries, the resulting reduction in the capillary bed and the increased resistance to the flow of blood, there is a rise in blood pressure. The tension then is increased throughout, and by constant irritation of the vessel walls and proliferation of the connective tissue, an arteriolosclerosis and later an arteriosclerosis of the vessel walls occur. That the changes in the vessels involve all the body tissues has been conclusively demonstrated by sections made of tissues from autopsies (Scott). It has been found that there is an arteriosclerosis, and sometimes an added arteriosclerosis of the kidneys, heart, brain and all the viscera. So far as limited clinical material permits the deduction (Mosenthal), an excess of sugar in the blood over short periods of time, and even over a period of several years, will not result in an increase in blood pressure. What relation the renal condition bears to the diabetes cannot be determined from the data available. Onfray (quoted by Gradle) found that 25 per cent of all diabetic patients have normal renal function and no hypertension; 50 per cent have hypertension and a partially intact renal function; 25 per cent have nephritis, with an excess of nitrogenous bodies in the blood.

The most surprising phenomenon is the length of time a moderate-sized hemorrhage will persist with little or no change. I have watched the same hemorrhage at intervals for a period of two months and have noted very little change. Iodides are being given to some patients with relatively large hemorrhages, with a speeding up of the absorption, but this offers no insurance against a new hemorrhage. When questioned because of the appearance of a new hemorrhage, a few of the patients have admitted the use of alcohol. New hemorrhages occur more frequently in persons who drink alcohol than in teetotalers. Calcium lactate is being given to a few of the patients whose vascular changes are not pronounced, but not enough time has elapsed to warrant an opinion as to its value in controlling the recurrence of hemorrhages. The advisability of the administration of calcium to patients who have marked vascular changes is questionable. There is every reason to believe that the larger vessels show more calcification in diabetic patients than in nondiabetic ones. Cammidge (1930), by roentgen examination of persons in the fifth decade of life, found that 36 per cent of nondiabetic patients and 63 per cent of diabetic patients showed calcification. Of the diabetic patients, 5 per cent had had the disease less than four years, and 50 per cent had had it for from five to ten years. Morrison and Bogan found that the degree of calcification increased with age and the duration of the disease.

SUMMARY

1. Diabetic retinitis is not a definite entity.

2. Most of the patients with diabetic retinitis who were studied had an arteriolosclerosis.

3. The majority of the patients under discussion had hypertension.

4. Improvement of the general diabetic condition is not always followed by improvement of the retinal condition.

5. Insulin accentuates the hemorrhagic condition of the retina when the patient has a reaction or shock.

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News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Paul Römer Festschrift.—The February number of the *Zeitschrift für Augenheilkunde* is devoted to a "Festschrift" in honor of the sixtieth birthday of Paul Römer, professor of ophthalmology in the University of Bonn. The number contains an excellent photograph of Professor Römer, and the following papers: 1. "Examination of Changes in the Permeability of the Vessel Walls in Enucleated Eyes of Rabbits, and Additional Attempts to Determine the Absolute Blood Quantity in Rabbits' Eyes with Radioactive Indicators," by Dr. K. Schmidt, Bonn. 2. "Central Scotoma in Diseases of the Chiasm," by Dr. E. Riegel and Dr. K. Vogelsang, Bonn. 3. "Nonoperative Treatment of Retrobulbar Neuritis," by Dr. E. Haas and Dr. K. Schmidt, Bonn. 4. "Perimetry," by Prof. F. Best, Dresden. 5. "*P*-butylaminobenzoyl *Di*-Methyl-Aminoethanol Hydrochloride," by Dr. H. Gebb, Frankfort. 6. "Trachoma," by Dr. E. von Grosz, Budapest. 7. "Actinomycosis of the Canaliculus," by Prof. W. Gruter, Marburg. 8. "The Frequency of Ocular Symptoms, Particularly Disturbances of the Pupillary Reaction After Syphilitic Infection," by Dr. K. von Hofe, Cologne. 9. "Quantitative Comparative Investigations on the Action of Local Anesthetics on the Corneae of Rabbits," by Dr. M. Kochmann and Dr. J. Nabe, Halle. 10. "The Etiology of Infantile Glaucoma," by Dr. Löhlein, Freiburg. 11. "Demonstration of the Lacrimal Passages with Thorium Dioxide," by Dr. W. Wegner, Freiburg.

Dog Guides for the Blind.—For the blind population of the United States unable to secure the direction of a companion, the provision of an educated dog guide will bring much freedom and independence. For four years, a nonprofit organization, The Seeing Eye Inc., Morristown, N. J., has provided approximately one hundred educated dogs for this work. The dogs are of the German shepherd breed, and are selected on the basis of intelligence and of responsiveness to training. The dog learns to match his gait to that of his master, and to give him direction. This is accomplished through the means of a harness and a U-shaped handle, which the sightless person holds in his left hand. In this way the dog protects the master from obstacles, transmits "up and down" signals for steps, and provides guidance through traffic. Since many of the blind know the environment well, considerable aid is secured from the dog, which will obey commands, such as "right," "left" and "forward."

Annual Colorado Summer Graduate Course in Ophthalmology.—The eleventh annual summer graduate course in ophthalmology at Denver will be given this year from July 17 to 22, inclusive. Special attention will be given to: (1) "Errors of Refraction, Their Measurement and Treatment"; (2) "The Cross-Cylinder, Cycloplegics and Form of Lenses; Contact Glasses"; (3) "The Ocular Muscles; Nonoperative and Operative Treatment of Squint"; (4) "Practical Fusion Training;

Prisms"; (5) "Biomicroscopy With the Slit Lamp, Instruments and Technic"; (6) "Surgical Procedures, Indications and Technic"; (7) "The Minute Anatomy of the Eye; Slides and Enlargements," and (8) "The Ocular Lesions of Tuberculosis." The Secretary, Dr. William M. Bane (1612 Tremont Street, Denver), will answer any inquiry for further information.

Institute of Ophthalmology, Columbia University.—On Jan. 16, 1933, without ceremony the Institute of Ophthalmology of the College of Physicians and Surgeons of Columbia University opened its doors. The plant and equipment were provided through the generosity of Mr. Edward S. Harkness. The Institute provides eighty-five beds, of which twenty-five are private, for the care of patients, facilities for undergraduate and postgraduate teaching of ophthalmology and well equipped laboratories for the investigation of problems relating to the eye. Dr. John M. Wheeler is the director of the Institute and the active head of the department of ophthalmology. Pathology is under the direction of Dr. A. B. Reese, bacteriology under Dr. Richard Thompson, chemistry under Dr. Karl Meyer and investigation under Dr. Howard Adelmann. Other members of the staff are: Dr. John H. Dunnington, Dr. Thomas H. Johnson, Dr. Daniel B. Kirby, Dr. Gordon M. Bruce, Dr. John P. Macnie, Dr. Hugh S. McKeown, Dr. Howard D. H. Brown, Dr. Carlos Finley, Dr. Manuel Uribe Troncosa, Dr. Newell W. Giles, Dr. Ramon Castroviejo, Dr. Raymond N. Berke, Dr. Laurence D. Redway, Dr. John W. Palmer and Dr. Edward Gallardo.

SOCIETY NEWS

New York State Medical Society.—A meeting of the Section on Ophthalmology was held on Tuesday, April 4. The chairman was Dr. David F. Gillette, Syracuse; the secretary, Dr. Frank M. Sulzman, Troy. The subject for discussion was "Chronic Simple Glaucoma," and the program as follows: 1. "Diagnosis," Dr. Searle B. Marlow, Syracuse. Discussion opened by Dr. Arthur J. Bedell, Albany; Dr. John N. Evans, Brooklyn. 2. "Pathology," Dr. Algernon B. Reese, New York. Discussion opened by Dr. Bernard Samuels, New York, and Dr. Harold H. Joy, Syracuse. 3. "Surgical Treatment," Dr. Arnold Knapp, New York. 4. "Medical Treatment," Dr. William Zentmayer (by invitation), Philadelphia. Discussion opened by Dr. Albert C. Snell, Rochester; Dr. Walter S. Atkinson, Watertown, and Dr. Thomas H. Johnson, New York.

Fourteenth International Congress of Ophthalmology.—The Fourteenth International Congress of Ophthalmology was held at the Palace Hotel, in Madrid, on April 16 to 22, 1933. The program was as follows: April 16. Inauguration at the University with the President of the Spanish Republic in the chair; inauguration of the scientific and industrial expositions; visit to "Ciudad Universitaria"; in the evening, a reception and dance at the Palace Hotel. April 17. Program of the International Association for the Prevention of Blindness; general communications; demonstrations; in the evening, a banquet. April 18. "Tuberculosis of the Iris and Ciliary Body," by Dr. E. V. L. Brown, of Chicago; "Pathology," by Dr. J. Igersheimer, of Frankfort.

Germany; "Diagnosis," by Dr. Henri Lagrange, of Paris; discussions and communications; in the evening, a reception at the National Palace. April 19. Standardization; discussion; excursion to the Escurial; in the evening, a reception. April 20. "Detachment of the Retina," by Prof. Ramon y Cajal; "Etiology," by Dr. H. Arruga, of Barcelona; "Medical Treatment," by Dr. G. Ovio, of Rome, Italy; "Operative Treatment," by Prof. A. Vogt, of Zurich, Switzerland. The afternoon and evening were devoted to pleasure trips and concerts. April 21. Program of the International Organization Against Trachoma; communications and discussions; visits to hospitals and ophthalmic clinics; closing ceremonies. April 22. Excursion to Toledo; banquet.

Forty-Sixth Congress of the French Ophthalmological Society.—The congress will take place in Paris, on June 26 and succeeding days. The principal report will be presented by Dr. V. Morax, on "Follicular Conjunctivitis." The secretary is Dr. René Onfray, 6 Avenue de la Motte-Picquet, Paris.

German Ophthalmological Society.—The next meeting of the society will take place at Heidelberg on Aug. 3 to 5, 1933.

Oxford Ophthalmological Congress—The twenty-third annual meeting of the congress will be held at Keble College, Oxford, England, on July 6 to 8, 1933. In addition to the Doyne Memorial Lecture by Mr. Bernard Cridland, there will be a symposium on the "Treatment of Glaucoma," a discussion on "Diathermy in Ophthalmology," with particular reference to its use in detached retina, by Mr. Affleck Greeves and a number of other interesting papers. Members desiring to contribute papers or demonstrations are asked to communicate with the secretary, Mr. C. G. Russ Wood, Hill House, Abberbury Road, Iffley, Oxford.

UNIVERSITY NEWS

Dr. Frederick Berg has been appointed professor in the University of Uppsala, and director of the University Eye Clinic, succeeding Professor Nordenson. The first occupant of this chair was Professor Gullstrand.

Dr. Louis Lehrfeld has been appointed neuro-ophthalmologist to the Wills Eye Hospital.

PERSONAL

The Swedish Medical Society has awarded the Gullstrand gold medal to Prof. Priestley Smith, of Birmingham, for his work on glaucoma.

DEATHS

Following a heart attack, Dr. George Slocum of Ann Arbor died suddenly, March 24. A graduate of the University of Michigan, Dr. Slocum since 1904 was connected with the department of ophthalmology at the University, and one year ago became head of the department.

We regret to announce the death, on April 4, of Mr. John Herbert Fisher, consulting ophthalmic surgeon to St. Thomas's Hospital and to the Royal London Ophthalmic Hospital.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

EXPERIMENTAL STUDIES AND CLINICAL EXPERIENCES WITH THE ANTIVIRUS OF BESREDKA. H. SCHMELZER, Arch. f. Ophth. **128:** 447 (June) 1932.

The author experimented with an antivirus obtained from a strain of Staphylococcus aureus (one made by Besredka himself and another made by a local bacteriologic institute). The virus was injected into rabbits subconjunctivally, resulting in the development of a conjunctival abscess of certain size which healed within a certain number of days. Injection of the antivirus two days before the inoculation with the virus did not alter the course of the formation of the abscess to any noticeable extent, with the only exception that in rabbits which had never come in contact with the specific antivirus larger abscesses usually developed than in the "immunized" eyes or their untreated mates. The antivirus used in those experiments hampered definitely, but without any specificity, the growth of staphylococci on blood-agar cultures.

The clinical experiences did not agree with the negative experimental findings as a few patients with chronic blepharoconjunctivitis with Staphylococcus aureus in the culture were definitely improved by the use of drops or salves containing the antivirus. The author used it in twelve patients (seven with chronic blepharoconjunctivitis, four with chronic conjunctivitis and one with pseudomembranous conjunctivitis). Five of the patients with blepharoconjunctivitis responded well to the antivirus; in the others the condition either was unchanged or became worse.

P. C. KRONFELD.

Blind

BLINDNESS IN SOVIET RUSSIA. A. S. SAVATIEFF, Sovet. vestnik oftal. **1:** 1 (Jan.) 1932.

The author gives the statistics of absolute blindness in Russia according to the general census taken in 1926. He considers the data not to be very accurate, as the census was taken by people who were untrained in ophthalmology. The author presents numerous tables embracing statistics of blindness according to sex, age, various nationalities and various regions of the vast country, also of acquired and congenital blindness. Out of the total population of 147,027,915 there were registered 234,833 absolutely blind persons, 101,874 men and 132,959 women. The incidence of blindness per hundred thousand of the population was 159.7; the incidence of acquired blindness was 139.55, and that of congenital 20.15, or 12.6 per cent of the total blindness, equally distributed between men and women. Of registered cases

of total blindness 11.1 per cent were among the urban and 88.9 per cent among the rural population. The incidence of blindness in the cities was 104.2 for males and 93.8 for females, while among the rural population the incidence was much higher for women than for men.

The author compares his data with those of Golovin in 1897, when the incidence of blindness was 197. The decrease of blindness at the present census to 159.7 the author ascribes to the prophylactic and educational measures spread among the population by the Soviet government.

O. SITCHEVSKA.

Cornea and Sclera

THE USE OF FASCIA LATA IN AN EXTENSIVE KERATOCELE. H. M. EMMONS, Am. J. Ophth. **14:** 1014 (Oct.) 1931.

A man, aged 40, was struck in the left eye with a piece of hot wood charcoal. Examination six days later revealed that the cornea had an almost central, circular area 6 mm. in diameter, dark gray and bulging. The tension was high, and there were ciliary injection, considerable hypopyon and some hyphemia. The cornea ruptured and sloughed, finally leaving a large hernia of Descemet's membrane. A button of fascia lata was laid in the defect and covered by a 9 by 22 mm. strip placed horizontally and fastened to the internal and external recti muscles. The upper lid was kept closed by a suture through it and fastened to the cheek by adhesive tape. A year later the eye was quiet, with vision of 20/70, and there was no bulging of the cornea.

W. S. REESE.

General

CATARACT AND OTHER OCULAR CHANGES IN VITAMIN G DEFICIENCY. P. L. DAY, W. C. LANGSTON and C. S. O'BRIEN, Am. J. Ophth. **14:** 1005 (Oct.) 1931.

Water-soluble vitamin B consists of an antineuritic element designated B or B_1, and a pellagra-preventive element G or B_2. Previous researches have shown that lack of the latter causes loss of the eyelashes, conjunctivitis and ophthalmia. In only one instance in the literature was opacity of the lens mentioned as a sequel. Young healthy albino rats from different litters and from stock unaffected with congenital cataract were given a diet deficient in vitamin G. Control animals from the same litters were given the same diet with the addition of autoclaved yeast to provide vitamin G. This group grew normally and showed no ocular or other pathologic condition. In 94 per cent of the rats on the vitamin G-deficient diet cataracts developed in from sixty to eighty-seven days. One rat in which a unilateral cataract developed showed no further changes in the lens when vitamin G was added to the diet. The authors give the pathologic findings in the lids and eyeballs removed at different stages.

W. S. REESE.

A Case of Filariasis Oculi. K. Koman Nayar and A. Kandaswami Pillai, Brit. J. Ophth. **16:** 549 (Sept.) 1932.

The patient was a police constable, aged 34, in Calicut. He came from a district where filariasis is fairly common, and his peripheral blood contained Microfilaria bancrofti. In the opinion of Wright, there has not been any case of intra-ocular filarial worm reported which has a greater claim to be regarded as bancrofti.

In the left eye, in the vitreous just in front of the macula, was seen a threadlike, wriggling worm, coiling itself and moving briskly. It appeared to be about 3 mm. long. Nineteen days later the worm was found lying in the bottom of the anterior chamber, and it moved about briskly when light was thrown on it. It was grayish white and was approximately 2.5 cm. long. The anterior chamber was opened with a keratome, and the escaping vitreous apparently carried the worm with it, but the worm was lost despite all precautions.

W. Zentmayer.

General Pathology

Histologic Examination of Neuritis Papulosa (A. Fuchs). Paulina Satanaowsky, Arch. de oftal. de Buenos Aires **7:** 279 (May) 1932.

This is a pathologic report of a case of the disease clinically described by A. Fuchs (*Ztschr. f. Augenh.,* 1926, vol. 59). The eye had been removed for a syphilitic iridocyclitis fourteen years previously.

The disease as described by Fuchs appears from four months to two years after a syphilitic infection, and in the reported cases, has been unilateral, after being preceded in the same or the other eye by iritis. The ophthalmoscopic findings are: a vitreous filled with fine and thick opacities; an exudative mass of a gray and yellowish-gray color situated at the optic disk, hiding its details and projecting into the vitreous, accompanied by numerous retinal and at times preretinal hemorrhages. Foci of choroiditis may also be observed, which subsequently give rise to patches of choroidal atrophy. The exudate extends along the blood vessels into the retina. The visual disturbance is great and lasts a long time. The field changes consist in a central scotoma corresponding to the exudate or an enlargement of the blind spot.

The histologic examination of the anterior segment shows a chronic iridocyclitis with secondary glaucoma and cataract formation. That of the posterior segment shows a leukocytic infiltration surrounding the blood vessels with hemorrhages, edema and fibrinous exudate in the thickness and on the surface of the retina. The cribriform plate is infiltrated and pushed backward, with retention in the depression of numerous lymphocytic elements circularly disposed about arteries obstructed by proliferating endothelium. In the papillary exudate, connective tissue is found in cordlike formation such as is found in proliferating retinitis. The papillary exudate extends over the retina between this and the fibrinous exudate, lining its internal surface and involving the retina, where lesions of an obliterating endarteritis are observed.

C. E. Finlay.

Glaucoma

CONCERNING CYCLODIALYSIS IN SIMPLE GLAUCOMA. H. S. GRADLE, Am. J. Ophth. **14:** 1101 (Nov.) 1931.

The author states that cyclodialysis has not received the consideration it deserves. Twenty-seven cases are considered from the following standpoints: (*a*) failures, (*b*) partial success, (*c*) success, (*d*) cataract formation and (*e*) increase in vision. The following conclusions are made: 1. Cyclodialysis offers practically 80 per cent chance of success, provided the indications for the operation are adhered to. 2. The indications for cyclodialysis in simple glaucoma are: (*a*) pure simple glaucoma without inflammatory reaction; (*b*) that type of case in which continued use of miotics just fails to maintain the integrity of the visual fields, the visual acuity and the balance of intra-ocular tension, and (*c*) the malignant simple glaucoma in which the peripheral defect of the visual field reaches to within 10 degrees of the point of fixation. 3. Cataract formation occurs in slightly more than 10 per cent of the cases after cyclodialysis. 4. When proper indications exist, cyclodialysis is to be preferred to iridectomy or fistulizing operations because of: (*a*) less damage to the integrity of the eye as a visual organ, (*b*) less danger of immediate postoperative complications, (*c*) no danger of late infection and (*d*) possibilities of repetition if unsuccessful.

W. S. REESE.

ABNORMAL SIZE OF THE LENS AS CAUSE OF JUVENILE GLAUCOMA. E. KURZ, Klin. Monatsbl. f. Augenh. **87:** 433 (Oct.) 1931.

The vision of a baker, aged 18, and previously healthy, was reduced to counting fingers at 1 meter in the right eye, and to 1.5 meters in the left; it could be brought up to 5/20 with minus 18 diopter lens in the right and to 5/15 with 16.0 in the left. The corneas were clear; their diameter measured 10.5 mm. The anterior chambers were narrow, especially in the center; the iris in both eyes was normal, and the vitreous was clear. The disks presented deep, but not marginal, excavations; intra-ocular tension was 45 mm. in the right eye and 47 mm. in the left. The marked enlargement of the lenses prompted the author to take exact measurements of their dimensions and locations, which data are given in detail. Compared with the figures obtained by Helmholtz, Herman Knapp, Donders-Horstmann and Raeder, the lenses were found to be greatly thickened and pushed forward; the radius of the corneas was nearly normal and their diameter was smaller than normal; the longitudinal axis of the globes was 5 mm. longer than normal. In this case of juvenile glaucoma the eyes presented a small anterior and an extended posterior portion, and a greatly enlarged lens. Taking the connection between the abnormal construction of the globes and the glaucoma as an evident fact, the author discusses the mechanism of the increase of the intra-ocular tension. Pilocarpine produced an acute glaucomatous attack with decided miosis, but it reduced the tension in the usual manner a month later. In the meantime iridectomy had been performed in both eyes, and communication between the anterior and posterior chambers was reestablished.

K. L. STOLL.

RESULTS OF HOLTH'S TANGENTIAL SCLERECTOMY AND IRIDENCLEISIS SUBCONJUNCTIVALIS IN GLAUCOMA SIMPLEX (1920 TO 1929). J. C. HOLST, Klin. Monatsbl. f. Augenh. **87:** 602 (Nov.) 1931.

The author renders a detailed report on, and digest of, the results obtained in simple glaucoma with Holth's tangential sclerectomy and subconjunctival iridencleisis at the University Eye Clinic at Oslo in the decade from 1920 to 1929. More than a dozen tables illustrate the material. The sclerectomy was generally combined with a small peripheral iridectomy, and only rarely with a total iridectomy since 1927. Patients with all stages of simple glaucoma, comprising vision of from more than 5/30 to the counting of fingers at 2 meters, and with a considerably contracted visual field, were operated on by these two methods. The observation was continued for a year or more after the operation.

After sclerectomy favorable tension without miotics was retained for at least one year after the operation in 81.9 per cent of 215 cases. Vision remained stationary, or was improved, in 59.3 per cent of this number. For a year or more after iridencleisis favorable tension without miotics was retained in 75 per cent of 81 cases, and stationary or improved vision in 86.3 per cent. The average figure for satisfactory tension and unchanged vision after sclerectomy was 54.1 per cent, and after iridencleisis 70 per cent. The best results were obtained with both methods when the eyes were operated on at an early stage of the disease. Cataracts occurred more frequently after sclerectomy than after iridencleisis, which explains why better visual results followed the latter method. Decrease of vision as a probable sequela to the formation of, or increase of, a previously existing cataract was found at least three months later in 20 per cent of the patients operated on by sclerectomy and in 7.5 per cent of those undergoing iridencleisis.

K. L. STOLL.

Hygiene, Sociology, Education and History

ACUTE GLAUCOMA: AN HISTORICAL NOTE. A. SORSBY, Brit. J. Ophth. **16:** 555 (Sept.) 1932.

While the term glaucoma goes back to Hippocratic writings, the conception of acute glaucoma is barely more than a hundred years old. It was not until 1813 that a really convincing description of acute glaucoma appeared—an account by Joseph Beers. An excellent description was given in 1818 by Demours. Rainbow colors and hardness of the eye are mentioned.

The first to realize the connection between arthritic ophthalmitis and glaucoma seems to have been Sir William Lawrence. He, like Weller, describes arthritic ophthalmitis and glaucoma as separate conditions. To him must be given the credit of introducing the term acute glaucoma.

Not until the conception of ocular hypertension and the findings of the ophthalmoscope had been applied could glaucoma simplex become a clinical entity.

It is to be hoped that this meager abstract may be of sufficient interest to cause the article to be read in full.

W. ZENTMAYER.

Injuries

BILATERAL TRAUMATIC PERFORATION OF THE MACULA. QUINT, Klin. Monatsbl. f. Augenh. **87**:(Dec.) 1931.

A man suffered a contusion of his right eye as a result of the explosion of a shell in 1915. A hole in the macula, one-fourth disk diameter in size, had remained unchanged since the accident. Vision was one twenty-fourth of normal. The left eye received a severe contusion from a piece of broken grindstone in June, 1931. After absorption of the hemorrhage in the vitreous, a hole in the macula was detected, one-half disk diameter in size. The hole was closed later by what appeared to be connective tissue, the patient recovering one twenty-fourth of natural vision.

K. L. STOLL.

Instruments

THE NEEDLE CAUTERY. E. H. OPPENHEIMER, Klin. Monatsbl. f. Augenh. **87**: 804 (Dec.) 1931.

The author has devised a new cautery, the one shank of which terminates in a finely pointed platinum needle, 4 mm. in length, which glows after from two to three seconds. The instrument can be used to advantage in epilation and cauterization of even the most minute corneal infiltrations and ulcers, with or without the aid of a slit-lamp. In Comberg's opinion, the needle cautery should be convenient in Gonin's operation and in puncturing the anterior chamber; also possibly in operations on keratoconus. It is manufactured by Wurach of Berlin.

K. L. STOLL.

Lacrimal Apparatus

CONCERNING LACRIMAL CYSTIC DIVERTICULI. MICHAIL, Arch. d'opht. **49**: 384 (June) 1932.

The author reports three cases of lacrimal cystic diverticuli. Two of the cases were congenital and one was traumatic in origin. The condition is rare, but has been recognized for many years. Most of these diverticuli have been described as having a prelacrimal location as in the third case reported by the author. In the first two cases the diverticuli were laterolacrimal in situation, which is exceptional. They may have a congenital origin or be the result of an inflammatory process.

S. B. MARLOW.

Lens

ANNULAR CONTUSION CATARACT OF VOSSIUS (CRITICAL STUDY). H. FRENKEL, Rev. cubana de oto-neuro-oftal. **1**: 154 (May-June) 1932.

Differing with the usual opinions, the author comes to the following conclusions:

1. The annular cataract of Vossius is nothing more than one of the symptoms of the traumatic syndrome of the anterior segment, and all cases described under this title are simply more or less characteristic examples.

2. The explanation given by Vossius of the mechanism of production of this cataract is, unlike that of Handmann, of value in regard to the pigmentation of the anterior capsule and the temporary swelling of the anterior layers of the lens.

3. This explanation shares with the classic theory the error as to the action of the contusing force by depression of the cornea on the iris and the iris on the lens, through an action from before backward, the error concerning the action of the aqueous humor.

4. In reality, one is dealing with a rupture of the zonula, a subluxation of the lens, an action of the lens on the posterior surface of the iris and a partial and temporary cataract, as in all cases of the traumatic syndrome of the anterior segment.

5. Thus, there is once more presented a fertile application of the general conception, which studies contusions of the anterior part of the globe in the light of the traumatic syndrome and not as isolated constituent symptoms.

S. L. RHODE.

Lids

THE TREATMENT OF SPASTIC ENTROPION BY THE INJECTION OF ALCOHOL INTO THE LID COMBINED WITH CANTHOTOMY. L. WEEKERS, Arch. d'opht. **49:** 427 (July) 1932.

Because of the relapses which he has observed following the use of the original technic described in 1927, Weekers has sought various means to intensify the action of this procedure, which he finds was first advocated by Fumazalli in 1909. To the 80 per cent alcohol he has added tincture of iodine and other drugs, and has also used repeated injections. Some recurrences have appeared in spite of these changes, but his results have been much improved by the technic he now employs. This consists first in the subcutaneous injection of procaine hydrochloride along the border of the lid and in the region of the outer canthus. The margin of the lid is then snipped 3 mm. deep with the scissors near the external canthus. The alcohol is then injected deeply under the skin to avoid injury to the skin.

When the spastic entropion is recent, associated with a painful ocular condition, injection alone is usually sufficient. When the entropion is of long standing and very annoying, canthotomy should be combined with the injection of alcohol.

S. B. MARLOW.

Neurology

ALTERATION OF CIRCULATION OF BRAIN BY SURGICAL MEANS IN DISEASES OF THE CENTRAL NERVOUS SYSTEM. N. D. ROYLE, Brit. M. J. **1:** 1063 (June 11) 1932.

Following a sympathetic thoracic trunk section for extensor spasm of one forearm, the author noticed that, while tone was decreased in the ipsilateral arm, the contralateral arm fell even more rapidly from the perpendicular position. Earlier workers had already shown that stimulation of the cervical sympathetic caused a constriction of the vessels in the brain with a decrease in the volume of the brain, and, since it is a well known physiologic fact that in the central nervous system hyper-

activity accompanies slight degrees of asphyxia or anoxemia (insufficient oxygen in the blood), it was felt that the reflex activity of the brain might be altered by varying its blood supply rather than by cutting peripherally directed rami of the sympathetic nervous system.

Several experiments of the author on goats demonstrated that tone could be decreased both in the normal and in the decerebrate animal by dividing the thoracic sympathetic trunk, the change in tone being manifested on the side opposite that on which operation was performed. When the skull and dura were opened, the cerebral vessels were found to be smaller, and anastomoses were less numerous on the side on which operation was performed.

The author describes the procedure he uses in man, the first thoracic sympathetic ganglion usually being removed. The ipsilateral effects consist of the usual signs of Horner's syndrome plus a diminution in the tone of the muscles on the same side of the face, upper limb and neck, which the author ascribes to decreased activity of the reflex arcs of the cervical cord and the facial nucleus, owing to the increased blood supply.

The contralateral effect is a decrease in tone of the whole contralateral side, which is less noticeable in the face. Rigidity, clonus and tremor, if formerly present, are also decreased.

Ipsilateral and contralateral effects are present. Sympathetic fibers of the cervical sympathetic and those going to the brain along the vertebral and carotid arteries are distributed to the arteries and veins of the brain. After cutting them, the skin of the face, head and upper limbs becomes pale, indicating a change in the arterioles and venules. The changes in cerebral vessels (in the goat) have been described. In human beings, Brearly found ipsilateral dilatation of the retinal vessels. The results obtained in disease are ascribed to the increased blood supply (decreasing the anoxemia), which is obtained by eliminating vasoconstriction. If a secondary cellular atrophy or sclerosis has occurred as a result of long-standing defective circulation, the possibility of restoration of function by altering the blood supply is remote.

In trigeminal neuralgia, which is probably due to hyperactivity of the gasserian ganglion as a result of vasoconstriction following toxemia of some kind, ipsilateral thoracic trunk section gave complete relief in four patients (without the possibility of a neuroparalytic keratitis).

In retinitis pigmentosa, relief is obtained if the patient retains central vision. The contracted fields are enlarged, and visual acuity is increased. If sight has been absent for years, there is no relief, owing to the retinal atrophy. Deafness was relieved in a patient also suffering from retinitis pimentosa, operation on the right relieving deafness on the left, and vice versa, although there were gaps in the scale. Headache was relieved in another patient who also had retinitis pigmentosa.

The operation also gave relief in various neurologic diseases in which there was rigidity, tremor or spasticity.

The procedure of sympathetic thoracic trunk section seems to offer some hope in early cases of retinitis pigmentosa. However, it must be remembered that the operation confers a Horner's syndrome on the patient.

W. F. DUGGAN.

MENINGIOMA OF THE SELLA TURCICA. MARX and BOEVE, Arch. d'opht. **49:** 417 (July) 1932.

The authors point out that meningiomas of the sella turcica are of interest to the ophthalmologist because they often produce symptoms referable exclusively to his field of activity. The optic nerve may be implicated in the orbit, in the optic canal, when the tumor arises from the meninges of the sella, or the olfactory fossa or the sphenoid bone. The symptoms accompanying these localizations are described, and the differential diagnosis between the types of optic atrophy is discussed. The earliest symptom which these cases present is diminished visual acuity, generally unilateral. Later, contraction of the field of vision occurs, first for colors and then for white.

The authors describe a case in which observation and operation were done by them. The patient was free from any symptoms except for loss of vision. There was marked temporal contraction of the field for colors, while that for white remained practically intact. This observation leads them to emphasize the necessity of examining the color fields when quantitative perimetry, as advocated by Cushing, cannot be carried out. The case described demonstrates that in spite of an atypical field of vision for white, the typical temporal contraction for colors led to the correct diagnosis in spite of the absence of all other symptoms.

S. B. MARLOW.

Ocular Muscles

PARALYSIS OF OCULAR MUSCLES IN DIABETES MELLITUS. A. SCHICK and J. SILBERMANN, Wien. klin. Wchnschr. **45:** 1255 (Oct. 7) 1932.

The authors report two cases in which paralysis of ocular muscles occurred owing to a diabetic condition. They call attention to the fact that diabetes may be the etiologic factor in spite of negative urinary findings. In Joslin's book such a condition is not mentioned; von Norden reported seven cases, while Dieulafoy found that paralysis of ocular muscles is a characteristic of diabetes and reported fifty-eight cases found in the literature. In the authors' first case, occurring in a woman of 58 years, the urine contained no sugar, but the blood sugar was 224 mg. per hundred cubic centimeters. Headache, double vision and paralysis of the external rectus muscle of the left eye subsided with ordinary diabetic care. The second patient, a man of 68 years, had a history of diabetes of long standing. His ocular condition consisted of double vision, complete oculomotor paralysis of the left eye and headache without involvement of other cranial nerves. The urine contained 2.2 per cent sugar, but the blood sugar tolerance curve was typical of severe diabetes.

L. L. MAYER.

Operations

A PRACTICAL AND ADVANTAGEOUS PROCEDURE IN OPERATING FOR SENILE CATARACT. A. TIERI, Ann. di ottal. e clin. ocul. **60:** 213 (April-May) 1932.

The advantages of making the incision with the keratome are the possibility of always avoiding the iris and of making a perfectly beveled

incision which heals promptly. The difficulties in making a large enough incision, however, have prevented many from using this method.

The author makes two incisions with scissors in the conjunctiva running diagonally, up and out and up and in from 11 and 1 o'clock. Undermining from each of these, two conjunctival pockets are formed over the lateral thirds of the future section. Then a medial limbal section 7 or 8 mm. long is made between the flaps with the keratome. With fine scissors this incision is prolonged 3 or 4 mm. at each side under the flaps. A peripheral iridectomy is usually performed, and the lens expressed after capsulectomy with forceps. The two flaps adhere to the sclera without sutures, closing the wound safely. S. R. GIFFORD.

OUR OPERATIVE TECHNIC FOR CATARACT BY "ELECTRODIAPHAKIA." J. LOPEZ LACARRERE, Rev. cubana de oto-neuro-oftal. **1:** 149 (May-June) 1932.

A new method of lens extraction has been used experimentally on animals (rabbits, pigs and sheep) for some months, and has been practiced successfully on several patients.

The modality employed consists of a small handle, called an "electrodiaphake," from the insulated glass end of which, on pressing a button in the handle, two very small steel wires (0.14 mm. in section) appear in bifid form. No pressure is made on the lens as the diathermic current makes the passage of these metallic threads into its tissues easy, at the same time producing a coagulation with resulting adherence to the electrode. The current is interrupted with a foot pedal, and the lens in the capsule is removed without counterpressure, with no difficulty and with a minimum of danger of loss of vitreous.

The diathermy tip is a modification of the "thermoflux A" of Siemens, so constructed that the intensity of the current can be measured and regulated before each operation.

The diathermy current does not produce coagulation of the vitreous even though the electrode comes in contact with it. Consequently, this method should make an ideal one for the removal of lenses that have become luxated posteriorly. Another important field for its use might well be in the extraction of the clear lens in cases of high myopia.

S. L. RHODE.

FIXATION OF THE GLOBE IN OPERATIONS. F. ROMEICK, Klin. Monatsbl. f. Augenh. **87:** 512 (Oct.) 1931.

The author has used the guide suture of Elschnig with perfect results for over ten years, also Goebel's forceps. His own method, however, which does not disappoint him in cases of brittle conjunctivae, seems to him the safer way. He inserts a suture through the conjunctiva and the external layers of the sclera close to the limbus, slightly below the temporal extremity of the horizontal meridian of either eye. The suture is inserted for a short distance only, then tied with a surgical knot and cut directly over it. This knot, caught with an ordinary fixation forceps, offers a firm grasp and will never tear out; it also allows the forward pulling of the globe and can readily be cut and removed after the operation. Romeick uses special needles, and he recommends Zade's piercing

needle (*Vorstichnadel*), an instrument employed with good result in strabismus operations (Zade: *Ber. d. deutsch. ophth. Gesellsch., Heidelberg*, 1930, p. 332).

K. L. STOLL.

Pharmacology

INTOXICATION WITH PHYSOSTIGMINE (ESERINE). H. DESOILLE, Presse méd. **40:** 1847 (Dec. 7) 1932.

Physostigmine (eserine) is an alkaloid of a very toxic nature. It is used as an internal medication in the sympathicotonies and tachycardia, and externally as a collyrium to cause miosis by excitation of the radial fibers of the iris sphincter, which in turn causes a hypotony of the globe. The author divides the intoxications into general and local. In the former, symptoms of abdominal pain, vomiting, dyspnea, muscular twitching and general discomfort are seen early and last a few hours. In very acute cases, grave symptoms are found. The vomitus is stained with bile or blood, lacrimation is increased, the heart is very rapid, and in fatal cases deglutition is difficult or impossible.

Locally, the salicylate in aqueous solution is irritating to the conjunctiva. With susceptibility or intoxication, the miosis is intense, pain is caused by cramped ciliary muscle, vision is blurred, and there is convulsive trembling of the palpebral muscles.

Treatment of the general intoxication consists of the use of apomorphine to help empty the stomach and the injection of 0.1 Gm. of spartine or from 6 to 10 mg. of lobeline intramuscularly. It may be necessary to give these intravenously and also to use artificial respiratory oxygen in combination with carbon dioxide. In the treatment of the local intoxication, the author advises against the use of atropine because the eyes are usually of a glaucomatous nature, and, instead, cold packs are found efficient. The pain may be relieved by antipyrine or acetylsalicylic acid.

L. L. MAYER.

PHYSICAL, CHEMICAL AND PHARMACOLOGIC QUALITIES OF EXTRACTS FROM THE LACRIMAL GLAND. D. MICHAIL and P. VANCEA, Arch. f. Ophth. **128:** 38 (Feb.) 1932.

The extract from the lacrimal gland lowers the frequency of the heart beat and makes blood vessels contract. When injected intravenously it lowers the pressure in the carotid artery and increases the cerebrospinal fluid pressure. It stimulates smooth muscles, especially the uterus and the tunica muscularis of the blood vessels. The extract speeds up the healing process of wounds of the skin and also the epithelialization of defects in the cornea (especially after instillation in the conjunctival sac).

P. C. KRONFELD.

Physiology

COMPARATIVE STUDIES ON THE PERIPHERAL AND CENTRAL RETINA: 4. TEMPORAL SUMMATION OF SUBLIMINAL VISUAL STIMULI AND THE TIME COURSE OF THE EXCITATORY AFTER-EFFECT. R. GRANIT and W. A. DAVIS, Am. J. Physiol. **98:** 644, 1931.

When an eye is exposed to short flashes of light, it has been found that the product of the intensity, *i*, and the time of exposure, *t*, are

constant at the threshold. The simple formula $it = c$ is valid for exposures between 2 and 30 sigmas. For very short periods the relation breaks down. The obvious analogy between this rule and the Bunsen-Roscoe law, according to which a photochemical reaction is directly proportional to the product of the time of exposure and the light absorbed, has led to attempts to correlate the two. As a result of this, it has been found by a number of authors (Hecht, 1929; Adrian and Matthews, 1928) that this constant is influenced materially by synaptic processes in the retina. Adrian and Matthews found that spatial summation exists between separated stimuli in the retina, and they proved that the interchangeability between area and intensity is due mainly to synaptic interaction.

Previous papers by Granit and Granit and Harper have shown that spatial summation between separated areas occurs also in the human eye.

The present paper records experiments to determine whether there is evidence for synaptic summation of two successive subliminal stimuli, in other words, temporal summation. The original paper should be consulted for the methods used. As a result of their experiments, which showed definite temporal summation in different parts of the retina and with different states of adaptation, the authors conclude that the absolute sensory threshold in the retina is ultimately conditioned by processes in the neuron; i. e., it is subjected to modifications by temporal, and probably also by spatial, summation. The process may take place at different levels in the retina, perhaps as early as in the receptor body or at the junction between the receptor and the bipolar cell. F. H. ADLER.

THE EFFECT OF TOTAL ABSENCE OF FUNCTION ON THE OPTIC SYSTEM OF RABBITS. LOUIS GOODMAN, Am. J. Physiol. **100**: 46, 1932.

A healthy, pregnant rabbit, near term, was placed in a totally dark room and allowed to give birth to her litter (of nine). Each day the mother was removed to the sunlight for two hours, but throughout the duration of the experiment, six months, the young were kept in total darkness. Elaborate precautions were taken to prevent any light from reaching the room.

When the young were 9 days old, and before their eyes were opened, three of the animals were removed to a dimly lighted room and the left eyes in two animals were removed under ether anesthesia. In the remaining rabbit the left eyelids were sutured together. The animals were then returned to the dark room, where recovery was uneventful. The rabbits were killed by asphyxia in the dark at varying intervals. One was transferred to the light at the end of five months for a study of its behavior and ocular reflexes. Three of the animals, including one on which enucleation was performed, remained in the dark for six months. All the animals were weighed, measured and examined closely for evidence of deficiency disease. The brains and eyes were immediately removed for gross and histologic study. A motion picture was taken of the behavior of the rabbit taken into the light.

A litter of rabbits born from the mating of the same buck and doe as used in the dark room experiments was raised in the light, and its members were killed at the same age intervals as the experimental rabbits. In addition, a number of control extirpation and canthoplasty experi-

ments were performed on very young animals raised in the light. In this manner the author has studied the results of excision of an end-organ, the partial elimination of function and the complete absence of function.

No gross differences were found in any part of the eyes, optic nerves or tracts, primary optic centers or occipital cortices. Histologic studies of the retina, optic nerves, superior colliculi, lateral geniculate bodies, pulvinar and cortex showed no changes from the normal. In other words, excluding the light from the eyes of these animals for six months caused no demonstrable changes in the gross or histologic structure of their eyes and visual tracts and centers. In the animals in which one eye was enucleated the usual changes due to degeneration were noted.

The lid and pupil reflexes of a dark room rabbit removed to the light after five months for a study of its reflexes and behavior responded immediately to the first stimulus of light. Within two weeks this animal showed excellent progress in its reactions to visual stimuli.

The author draws the following conclusions: Complete lack of optic stimuli in rabbits from birth to 6 months of age results in no discernible changes in the visual pathways, does not prevent the lid and pupil reflexes from responding to the first stimulus of light or the animal from quickly learning to respond to visual stimuli in regulating its behavior; nor is there any indication that the presence of the peripheral sensory visual field is a factor in the development and differentiation of the optic system in rabbits, or in the ability of that system to function.

F. H. ADLER.

THE RATE OF FORMATION OF CEREBROSPINAL FLUID IN ETHERIZED CATS. L. FLEXNER and H. WINTERS, Am. J. Physiol. **101:** 697, 1932.

A water-tight block was established at or rostral to the cerebellar peduncle, so fluid could not escape into the subarachnoid space via the foramina of Luschka, or past the block into the region of the cisterna magna.

The authors found that the best way to accomplish this was with a catheter fitted into a rubber balloon shaped to the contour of the parts. These balloons were made according to the method of Reynolds and Friedman (*Am. J. Physiol.* **94:** 696, 1930). The experiments reported in the present paper were made on adult cats under light ether anesthesia. The average rate of flow of cerebrospinal fluid from the aqueduct of Sylvius was 12 cc. per day. The rate in a series of thirteen cats varied between 9 cc. and 16 cc. of fluid per day. These differences could not be explained by a difference in body weight or in the weight of the brain or choroid plexus of the animals.

F. H. ADLER.

RESPONSE OF THE NICTITATING MEMBRANE TO PROLONGED STIMULATION OF THE CERVICAL SYMPATHETIC. OSCAR ORIAS, Am. J. Physiol. **102:** 87, 1932.

The author stimulated the preganglionic and postganglionic fibers of the cervical sympathetic nerve in cats, and observed the effects on the

contraction of the nictitating membrane. Prolonged stimulation of the preganglionic fibers resulted shortly in a fatigue of the muscle in the nictitating membrane.

Prolonged faradic stimulation of the preganglionic fibers resulted in a gradual decline in the height of contraction of the nictitating membrane. When the postganglionic fibers were stimulated under the same conditions, the height of the contraction was maintained. The relaxation observed when the preganglionic fibers are stimulated is not true fatigue, for the membrane contracts again if the frequency of the excitation is decreased. This "pseudofatigue" is probably related to the Wedensky inhibition. The author believes that its presence only during preganglionic stimulation can be explained by the long refractory period of the superior cervical ganglion. (See Bishop, G. H., and Heinbecker, P.: *Am. J. Physiol.* **100:** 519, 1932.)

F. H. ADLER.

THE ACTION OF INSULIN ON THE OCULAR TENSION. BISTIS, Arch. d'opht. **49:** 375 (June) 1932.

After briefly noting some of the reported instances in which the thyroid, hypophysis and gonads have had some definite relation to the intra-ocular tension, the author reviews some of the work reported by Richter on the effect of insulin on the intra-ocular tension. Richter has observed an acute attack of glaucoma in a diabetic patient under treatment with insulin. Poos has observed the intra-ocular tension to rise from 26 to 50 mm. of mercury after the injection of 15 units of insulin. The author reports two cases in which he has watched the intra-ocular tension following the administration of insulin. In one there was a slight rise in the intra-ocular tension, the blood sugar falling from 170 to 120 mg. In the second case no change occurred in the intra-ocular tension, the blood sugar falling from 330 to 250 mg. The first case is in accord with known clinical facts and the experimental work of Richter. The second case Bistis explains by the fact that the blood sugar was not sufficiently lowered so that osmotic and colloidal changes were brought about to influence the intra-ocular tension. These observations lead the author to point out that a diabetic person may have glaucoma, and that, therefore, the intra-ocular tension should be noted before insulin is administered.

S. B. MARLOW.

Retina and Optic Nerve

CYANOSIS RETINAE. P. C. KRONFELD, Am. J. Ophth. **14:** 1108 (Nov.) 1931.

A youth, aged 18, was observed for two years. He had shown a blue discoloration of the skin since he was 2 years old. At the age of 5 it was noticed that he became very short of breath on exertion. He was never able to walk upstairs. Death occurred at the age of 19 from cardiac failure. Examination showed marked cyanosis of the skin and visible mucous membranes, clubbing of the fingers, and a systolic blood pressure from 85 to 95. The eyes were slightly proptosed, and the conjunctival and anterior ciliary vessels much wider and darker than normal. The disks were purple red and swollen from 2 to 3 diopters. The

entire eyeground was of a dark purplish color, and the whole vascular tree was enormously widened, especially the veins. There were no hemorrhages. Vision was 6/6 in the right eye and 6/18 in the left, and the ocular movements, accommodation and visual fields were normal. The patient stated that up to his sixth year the left eye had turned in. A complete autopsy report is given, including observations on the heart, viscera (which showed situs inversus of the intestines) and the eye.

W. S. REESE.

THE POSSIBLE RÔLE OF POLYPEPTIDEMIA IN THE CAUSATION OF RENAL RETINITIS. DEJEAN, Arch. d'opht. **49:** 446 (July) 1932.

Two cases are described in which chemical investigation has thrown suspicion as a possible toxic agent in the causation of albuminuric retinitis on the polypeptides of the blood. The first case was that described by Puech and Cristol. This patient when first observed had an albuminuric retinitis without evidence of nitrogen retention but with an elevated polypeptide index. A year later nitrogen retention was demonstrated, and the patient died in a short time. Thus, in this case, the retinitis and the increased polypeptide index were better signs of an impending uremia than the amount of urea in the blood.

The second case was first described by the author in conjunction with Villard and Cazals. It is presented in detail. The patient was found to have a characteristic albuminuric retinitis. Clinical examination was entirely negative, except for a high blood pressure and a high polypeptide index in the blood. In the discussion of these two cases the author points out that the term albuminuric retinitis leads to a false conception and should therefore be rejected. There is no parallelism between it and albuminuria, and it does not occur in acute nephritis with edema. He reviews briefly hypercholesteremia which has been incriminated as a cause of the retinal changes. He calls attention to the work which has been done on the "residual nitrogen" as the cause. From the evidence available, it would appear that the polypeptides have a severe toxic action, and that the polypeptide index is always a more exact indication of the severity of the clinical condition than the amount of urea. A consideration of the facts known leads the author to suggest that further research may prove this agent to be the cause of the retinal changes. The two cases observed seem to support this possibility.

S. B. MARLOW.

RETROBULBAR NEURITIS OF NASAL ORIGIN. E. VON HIPPEL, Arch. f. Ophth. **128:** 23 (Feb.) 1932.

The essence of this paper is von Hippel's reply to an acute question of the present day: Is the improvement of the retrobulbar process that frequently follows surgical procedures on the sinuses a post hoc or a propter hoc proposition? Are the "successes of the nasal therapy" actually the result of nasal operations, or are they produced by spontaneous recovery? Von Hippel believes the latter to be the case. He bases his conclusions on a new series of thirty-six cases (in addition to his first series of seventy cases), in thirty of which there was no pathologic condition in the sinuses on thorough roentgen examination.

The remaining six patients whose sinuses were not investigated roentgenologically presented definite cases of multiple sclerosis or hereditary optic atrophy. Twenty-one of the thirty-six patients were cured, in ten the condition showed an unfavorable course and in five the final outcome could not be determined. In the ten unfavorable cases operative procedures were, a priori, out of the question because of the underlying definite general pathologic conditions (multiple sclerosis, hereditary atrophy, myelitis). No patient was operated on. If operations had been performed in every case, the surgical method of treating retrobulbar neuritis would again have scored 70 per cent successes. The same consideration applies to the endonasal epinephrine tampon treatment. Hereditary optic atrophy is surprisingly frequent in von Hippel's material.

P. C. KRONFELD.

HISTOLOGIC EXAMINATION OF AN EYE AFTER GONIN'S OPERATION. BÖMER, Klin. Monatsbl. f. Augenh. **87**: 528 (Oct.) 1931.

An eye was enucleated two weeks after the second cauterization according to Gonin's method, when a fresh detachment of the retina aroused suspicion of the presence of a tumor. Histologic examination revealed no trace of a tumor. Broad and firm adhesion of the retina at the site of the ignipuncture could be observed in the microscopic sections. The sclera was necrotic at the site of the puncture, and its channel through the sclera was filled with granulation tissue in which numerous giant cells could be seen.

K. L. STOLL.

PROGNOSIS IN RETINITIS ALBUMINURICA (ANGIOSPASTICA). W. HEIDER and O. LÜRMANN, München. med. Wchnschr. **79**: 1585 (Sept. 30) 1932.

Under the conception of retinitis albuminurica have been grouped, previously, all of the retinal diseases due to acute and chronic nephritis, secondary contracted kidney and primary nephrosclerosis with or without loss in renal function. Lately there has been an attempt to differentiate retinal changes according to the nature of the basic disease. The authors review the several theories as to the physicopathologic production of retinitis. Volhard considers the arteriospastic changes in the retina and in the kidney as coordinate phenomena. Schieck feels that the lighter forms of retinitis are found both in the essential hypertensive type and in the nephritic type, due primarily to edema.

Moore and also Fishberg differentiate arteriosclerotic and so-called malignant hypertensive retinitis by the quantity and distinctness of the hemorrhages and exudates. Wagener attempts to separate the malignant hypertonic retinitis of chronic nephritis from the malignant hypertensive retinitis but finds little to differentiate. Guist finds that the peripheral venules of the retina show a tortuosity in primary hypertension, essential hypertension and malignant nephrosclerosis which is seldom found in the secondary hypertension of chronic nephritis.

The finding of these retinal conditions has, in the past, been a *signum mali ominis* as far as prognosis is concerned, as shown by the works of Fuchs, Leber and others. In acute nephritis and eclampsia recovery is the rule.

In two types called by the authors arterial ischemia of the retina and chronic pseudo-uremia recovery is the rule. They believe that prognosis is better gaged by the response of the arterial hypertension to treatment than by the retinal appearance. The bibliography is extensive.

L. L. MAYER.

MORPHOLOGY OF NEUROGLIA OF OPTIC NERVE AND RETINA ACCORDING TO METHODS OF THE SPANISH SCHOOL. CHADGI OMAR BULATCH, Russk. arch. oftal. **8:** 305, 1931.

The article presents a thorough study of the subject. The comparable value of various methods of staining of the neuroglia is discussed. The technic of del Rio Hortega's sodium-silver method and that of Ramón y Cajal's impregnation of the tissue with gold-sublimate and ammonium silver-pyridine is given in detail.

In order to verify the findings of the Spanish school, the author studied the structure of the normal neuroglia of the visual apparatus according to the fundamental work of Marchesani. He used human, bull's, sheep's and rabbit's eyes. Most of his material consisted of bull's eyes, as fresh human material could not be obtained, and cadaver's eyes would show postmortem changes too soon. The use of bull's eyes did not influence the value of the work since the histologic pictures of human and bull's neuroglia resemble each other closely. Numerous excellent photomicrographs are given. The author suggests the addition of a small amount of hydrochloric or acetic acid to the fixation fluid to prolong the fixation time. He succeeded in finding the debatable Müller's supporting fibers with Hortega's method and the astrocytes with Ramón y Cajal's technic.

The author believes Hortega's method to be superior to all other Spanish methods because of its simplicity. None of the Spanish methods, however, has established definitely the disputable and complicated structure of the neuroglial tissue of the optic nerve and retina.

O. SITCHEVSKA.

Uvea

AN UNUSUAL RESULT FOLLOWING TRAUMATIC IRIDO-CYCLITIS. D. J. WOOD, Brit. J. Ophth. **16:** 546 (Sept.) 1932.

The patient presented iridocyclitis in each eye following extraction of a cataract. The right eye remained irritable and was enucleated. Clinically, the cornea was fairly bright, allowing new-formed vessels on the iris to be plainly seen. The biomicroscopic examination gave no evidence of the pathologic condition.

On the entire back of the cornea was a thick layer of new tissue, thinnest at the edge, but in the center reaching a thickness of two-thirds that of the cornea. The deepest parts next to Descemet's membrane were well formed layers of fibrous tissue; the more superficial layers were more granular in texture, containing fewer nuclei but more particles of uveal pigment and many blood vessels. There was no layer of endothelium.

The interesting feature is the great transparency of the tissue, which did not reveal itself clinically. The thickness of the light track through

the cornea was not noted when the examination with the biomicroscope was made. Histologically, the source of the fibrous deposits was a finely granular coagulum in the angle of the anterior chamber. There was no evidence of sympathetic ophthalmitis.

The author suggests that if the conditions present in this case could be imitated one might find clear corneal scars. For this purpose, he proposes the use of a contact glass in corneal ulcers. (Illustrated.)

W. ZENTMAYER.

Sympathetic Ophthalmia

SYMPATHETIC OPHTHALMIA OR SYPHILITIC IRITIS? R. SCHEERER, Klin. Monatsbl. f. Augenh. **88:** 41 (Jan.) 1932.

A man, aged 41, suffered a contusion and rupture of the right eye in November, 1930, followed by immediate amaurosis and slow recovery. Pain and inflammation, which recurred in the eye on Sept. 25, 1931, was interpreted as a sympathetic ophthalmia, and the eye was enucleated the following day. Slight irregularity of the narrow pupil of the left eye was observed; the reaction, however, was prompt, and vision was normal on October 15. Violent iritis developed two weeks later, with deposits on Descemet's membrane, an opaque vitreous and a broad posterior synechia. The diagnosis of sympathetic ophthalmia, based on the clinical and pathologic condition of the enucleated eye, was ruled out after further observation for these reasons: 1. The inflammation of the left eye did not arise before the thirty-sixth day, while only nine cases are known in which the second eye became involved in four weeks. 2. There was only a single nodule visible at the pupillary margin, while nodules usually occur in a larger number in sympathetic ophthalmia. 3. An atrophic area could be noticed with the slit-lamp next to this solitary nodule, indicating a preceding similar inflammation. 4. The Wassermann reaction was strongly positive; one injection of neoarsphenamine cleared up the posterior synechia immediately, and a course of injections of arsphenamine cured the iritis promptly. No syphilitic symptoms had been observed except a "measles-like rash" two years previously. The course that this case took again demonstrated that sympathetic ophthalmia is unlikely to develop after a certain limited space of time, and also that slight pathologic changes may be overlooked during an active inflammation, simulating sympathetic ophthalmia in a case of injury.

K. L. STOLL.

Therapeutics

BLINDNESS FROM METHYL ALCOHOL TREATED BY LUMBAR PUNCTURE. G. H. MATHEWSON and B. ALEXANDER, Canad. M. A. J. **26:** 679 (June) 1932.

Mathewson and Alexander report a case of methyl alcohol poisoning in which the blindness was successfully treated by lumbar puncture. From a consideration of all the facts, they advise that in this condition the following treatment be instituted as soon as possible: (1) repeated gastric lavage during the first four or five days to remove the alcohol excreted by the gastric glands; (2) a large fluid intake to dilute the

poisons circulating in the blood stream; (3) pilocarpine sweats and hot packs daily to aid elimination through the skin, and (4) what is most important, the withdrawal of as large an amount of cerebrospinal fluid as possible daily, for four or five days. The pressure should not be allowed to fall below 100 mm. of water, to dilute the poisons and mitigate their attack on the central nervous system, especially the eye. This treatment, of course, is to be used whether the eyesight is damaged or not.

EDITOR'S ABSTRACT [J. A. M. A.]

VACCINOTHERAPY IN TRACHOMA AND OTHER OCULAR INFECTIONS. V. RUATA, Ann. di ottal. e clin. ocul. **60:** 327 (April-May) 1932.

The author reviews Besredka's theory of local immunity and the result of attempts to apply it in ophthalmology, especially by the use of "antivirus." Since he believes that most of the complications of trachoma are due to infection with secondary organisms, he attempted to produce local immunity to all of the organisms likely to be found in the conjunctival sac by the use of a polyvalent vaccine containing killed organisms and filtrates of broth cultures supposed to contain antivirus. Results with the latter preparation were variable, and the reaction from subconjunctival injection was marked, so the mixed vaccine containing 5,000 organisms per cubic centimeter was used in most of the cases reported.

Animal experiments showed that both instillation and subconjunctival injection of the vaccine protected rabbits against inoculation with organisms which caused purulent conjunctivitis in controls, and that such protection lasted for at least twenty days.

Case reports and a table containing data on sixty-eight patients treated with the vaccine are given. These included fifty-seven cases of trachoma, most with corneal complications, three of gonorrheal ophthalmia, three of acute mucopurulent conjunctivitis (Koch-Weeks) and two of subacute conjunctivitis. When the symptoms and complications attributed to the secondary infections in trachoma are considered, 55 or 80 per cent were cured, the trachoma itself showing marked improvement or even complete cure during treatment.

From four to twenty-five subconjunctival injections or local applications were given each patient, the injections being usually given every two days and the applications daily. Ten cases which had resisted other treatment cleared up rapidly on the vaccine, forty-two cleared up with unusual rapidity and four in about the same time as other cases in which the vaccine was not employed. Of the thirteen refractory cases, most were of the scrofulous type or showed other signs of poor general condition. The author believes that recurrences of the complications may be prevented by resuming the vaccine treatment for two or three days every two or three weeks.

S. R. GIFFORD.

THE REACTION OF THE CORNEAS OF SYPHILITIC RABBITS TO THE INTRACORNEAL INJECTION OF FOREIGN PROTEIN. VON HOFE and KRANTZ, Arch. f. Augenh. **105:** 721, 1932.

A series of rabbits whose corneas were infected with Spirochaeta pallida were given an intracorneal injection of inactive horse serum. A

similar injection of horse serum was made into the corneas of non-syphilitic rabbits. A comparison of the two series of animals showed that the syphilitic rabbits gave a much more severe reaction to the injected serum than the normal controls.

F. H. ADLER.

TREATMENT OF OCULAR DISEASES WITH SODIUM HYDROCARBONATE. E. LUZSA, Klin. Monatsbl. f. Augenh. **87:** 769 (Dec.) 1931.

Pellathy and Schneider used sodium hydrocarbonate in the treatment of ocular diseases for the purpose of converting the pathologic acid reaction of the diseased tissues into a normal reaction (*Klin. Monatsbl. f. Augenh.* **85:** 774 [Dec.] 1930). Luzsa repeated these experiments, and found it impossible to demonstrate a diminution of the p_H values in the diseased eye, as compared with the healthy eye. He found, however, that the lymph of the tissues shows actually an increased acid reaction during inflammatory processes. Cases of trachoma were chiefly used for his research. The disappearance of pannus after protracted treatment with sodium hydrocarbonate, however, may also result from other therapeutic procedures. Ethylmorphine hydrochloride, in Luzsa's opinion, is an equally effective adjuvant in these cases, as is sodium hydrocarbonate. An alkali substratum, produced by this preparation, may even prove detrimental when many virulent germs are present. He quotes Sauerbruch's opinion, that the development of an acid reaction during a pathologic process may render the substratum less favorable to the growth of the bacteria. Sodium hydrocarbonate offers no therapeutic advantage, and may even cause irritation.

K. L. STOLL.

IONTOPHORESIS IN OCULAR DISEASES. S. D. OSIMIN, Russk. j. oftal. **14:** 301 (Oct.-Nov.) 1931.

The author describes the construction of various electrodes and their use in ophthalmology. He used Cantonnet's electrode, increasing the strength of the current gradually from one-fifth to two milliamperes each séance. Zinc sulphate, 1:1,000, sodium iodide, sodium salycilate and potassium chloride 1:400 served as electrolytes. From three to thirty treatments were given daily from fifteen to thirty minutes to forty-one patients, or to forty-nine eyes. In thirty-three eyes the cornea was affected, twenty having ulcers. The other diseases treated were vitreous opacities, glaucoma, episcleritis and cataract. The best results were obtained in corneal ulcers in which the treatment stopped the progress of the disease and led to regeneration of the tissue with clearing of the opacities. Iontophoresis proved to be a favorable aiding factor in vitreous opacities and in recent leukoma of the cornea. In glaucoma, the effect was not lasting.

The author believes that iontophoresis should become more popular in the treatment of various diseases of the eye.

O. SITCHEVSKA.

BLINDNESS DUE TO ANGIOSPASM OF THE RETINA CURED BY RETROBULBAR INJECTIONS OF ATROPINE. A. TICHOMIROFF, Sovet. vestnik oftal. **1:** 98, 1932.

A girl, aged 18, became blind in both eyes within two days. The pupils were dilated and did not react to light and in convergence. The

fundi presented a typical picture of embolism of the central artery, the cherry red spot being present in the macular region of both eyes. Retrobulbar injections of 0.1 per cent solution of atropine sulphate were given in quantities of from 0.3 cc. to 1 cc. four or five times. The vision was improved to 0.5 in the right and to 0.7 in the left eye within a month. The cherry red spots disappeared, but the optic nerves remained pale.

The author believes that the blindness was due only to an angiospasm of the retina, because of the youth of the patient, the bilateral process and the absence of lesions in the internal organs and because of the favorable end-result.

O. SITCHEVSKA.

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RECENT DEVELOPMENTS IN INVESTIGATIONS ON TRACHOMA. DR. HARVEY J. HOWARD, St. Louis.

There seems to be an agreement among those most familiar with the clinical aspects of trachoma that the disease is always associated with conjunctival follicles which become confluent, producing a general lymphadenoid hyperplasia; also that sooner or later pannus develops in all cases of trachoma, a symptom that definitely establishes the diagnosis.

Peter and others have demonstrated in early trachoma the presence of elastic lattice fibers around the follicles and beneath the conjunctival epithelium. Peter concludes that these fibers, which are known to occur in sclerotic processes of lymph glands and the liver, are responsible for the scar tissue so typical of advanced trachoma.

The question of "trachoma bodies" cannot be dismissed on the assumption that they are identical with the epithelial cell inclusions induced by such irritants as nicotine, alcohol and croton oil. Some investigators have shown that certain irritants bring about an increase in the chromatin of the nucleus in consequence of which the cell, in its effort to balance the nucleocytoplasmic ratio, is forced to extrude nuclear material into the cytoplasm. This material often resembles the cap of trachoma inclusions, but their staining reactions are different.

The suggestion that trachoma is a disease of nutritional deficiency is based almost entirely on clinical observation. It is possible that lack of vitamin A lowers the resistance of ocular mucous membranes to infection.

Evidence is still lacking that trachoma is caused by a filtrable virus.

The results of bacteriologic studies on trachoma indicate that the basal flora is not particularly characteristic and runs fairly constant. The bacteria isolated most frequently have been staphylococci and diphtheroids, but it is difficult to attach much significance to these findings. Organisms have been isolated from such cultures, which have been advanced as the cause of trachoma, but sufficiently conclusive evidence has not yet accompanied any of these claims.

The recently discovered Bacterium granulosis is considered by some as possessing the greatest possibility as the cause of trachoma. While Noguchi and Olitsky have isolated this organism in a number of trachomatous cases, and while in their hands it sometimes induces a

conjunctival reaction in monkeys which clinically simulates the conjunctival reaction in human trachoma, nevertheless the great majority of workers have been unable to isolate it or to produce definite lesions in the monkey's conjunctiva following the injection of fresh cultures of the organism. Furthermore, all experiments to induce trachoma in human subjects by the injection of Bacterium granulosis have thus far been uniformly negative (with the exception of one doubtful case). Recent communications report that the organism has been recovered from cases of folliculosis. The evidence in favor of Bacterium granulosis is therefore steadily becoming weaker.

DISCUSSION

DR. MARTIN COHEN, New York: When, in May, 1927, Dr. Noguchi presented before the American Medical Association his findings on Bacterium granulosis, I discussed the paper, and at that time I reached the conclusion that he had isolated a micro-organism capable of producing in monkeys and chimpanzees a follicular conjunctivitis similar to that brought about by a direct transmission from man to monkeys. I am still of this opinion, in the absence of further evidence to the contrary in regard to conjunctival cicatrization and pannus. Whether the condition as produced in animals is the same as human trachoma has not as yet been proved. I understand from Dr. Olitsky that Bacterium granulosis has been absent in the six cases of follicular conjunctivitis that he has studied, and was also absent in five patients studied by Biette. The inclusion cells of Prowazek and Halberstaedter, sometimes called trachoma bodies, were also considered originally to be the cause of trachoma. They were recovered in trachoma, rarely and with difficulty, from conjunctival scrapings in epithelial cells stained with Giemsa's solution.

The hypothesis that the inclusion cells are responsible for an independent conjunctival disease cannot be absolutely proved until a means is discovered of growing these bodies in pure culture. There is, however, sufficient evidence that the term trachoma bodies is a misnomer and should be discarded.

I have had no personal experience with the filtrable virus of trachoma and Bacterium granulosis, or with a diet deficient in vitamin A. However, I believe that diet, as mentioned by Dr. Howard and others, has no direct influence in the causation of trachoma or the presence of Bacterium granulosis.

DR. SANFORD R. GIFFORD, Chicago: The inclusions we produced in the epithelial cells of animals following the instillation of nicotine and croton oil were considered morphologically indistinguishable from those found in trachoma. Although inclusions could be found in a number of animals so treated, they were never numerous, only one or two typical ones being found, as a rule, in a cover-glass preparation. Ozuchi and Mikuchi, in 1930, reported the use of a 2 per cent infusion of a drug containing saponin to produce an increased mucoid secretion in suspected cases of trachoma, inclusions being especially numerous, according to these authors, in such secretions. This infusion was used in the eyes of twenty-six infants, several days old, who had had no

previous discharge. The reaction was not severe and subsided within from twenty-four to forty-eight hours. Scrapings made during this time, however, showed perfectly typical inclusions in three cases. The preparations were shown to a number of ophthalmologists, including Dr. Wilson, of Cairo, and the members of our department of bacteriology. These gentlemen considered the inclusions produced in the babies indistinguishable from those in trachoma. Professors Lindner and Löwenstein believed that there were differences in appearance, the exact nature of which was never made clear to us.

If these findings mean anything, it is that inclusions are the products of cellular reaction to certain agents, bacterial or chemical, which produce inflammation. Certainly the bodies in our preparations are not parasites, and it seems unlikely that the similar bodies found in trachoma, gonorrheal ophthalmia, swimming pool conjunctivitis and nongonorrheal conjunctivitis of the new-born are parasites.

Dr. S. Hanford McKee, Montreal, Canada: While no one can deny for a moment that trachoma is a contagious disease, I am under the impression that it is not nearly so contagious as we have been taught to believe. I have had very little to say about Bacterium granulosis, because I have felt I might be misunderstood; however, I believe that Bacterium granulosis is one of the large group of saprophytes found in the conjunctiva.

Photographed trachoma bodies are rather different from trachoma bodies seen under the microscope. I mean by that, that when you put a series of slides on the screen, the trachoma bodies, both the true and the so-called pseudobodies, appear absolutely similar. On the other hand, under the microscope, the slides appear to be different.

Dr. Harvey J. Howard, St. Louis: One thing I did not have time to say is that I do not consider trachoma more than mildly contagious. It certainly is not contagious to the same degree that inclusion blennorrhea is contagious. The example to which I referred was given for the purpose of showing that trachoma is a transmissible disease. Perhaps Professor Fuchs' case was unusual, but there are a number of cases on record which demonstrate that trachoma has been directly transmitted from one human being to another. While I do not say that inclusion bodies are parasites, still I am convinced that their presence in specimens must be considered as significant. In my paper I state that Taborisk found them in 163 of 164 cases of initial trachoma, and that in Peiping, China, my assistants and I found them in 65 per cent of all cases, both early and late. These percentages have largely been confirmed by several other workers.

An Unusual Form of Keratitis (Superficial Parenchymatous Punctate Keratitis). Dr. W. Thornwall Davis, Washington, D. C.

This form of punctate keratitis involves the superficial parenchyma, but not the epithelium. It is characterized by periodicity and symptoms of ocular irritation, with or without conjunctival congestion. It is seen only with the slit-lamp, the author using fluorescein stain and a magnification of twenty-eight times. Eight of the eleven cases on which

this paper is based were toxic in origin. The entity explains one cause for the frequent complaints of red eyes with lacrimation and concomitant symptoms of ocular irritation resembling attacks of conjunctivitis.

DISCUSSION

DR. WALTER E. CAMP, Minneapolis: On the basis of the changes found in the iris, the character of the corneal spots and their location around the corneal nerves, where they pierce Bowman's membrane to reach the epithelium, Verhoeff concluded that this type of superficial punctate keratitis is due to the action of a toxin on the ganglion cells of the ciliary ganglion. The location of these spots near the center of the cornea, rather than near the limbus, would substantiate this view. The hyperesthesia of the cornea, the lowered intra-ocular tension and the staining of these subepithelial spots with fluorescein would tend to classify this disease with the group of neuropathic or trophic disturbances of the cornea.

The clinical histories in the cases described by Dr. Davis are similar to those in cases of ordinary superficial punctate keratitis. There is in both types a history of focal infection or acute respiratory infection. In both the disease may be bilateral. In both there are chronicity and a tendency to acute exacerbation. In the objective examination, however, the spots differ in size and apparent location, although in ordinary superficial punctate keratitis many of the spots are extremely small and do not cause any disturbance of the epithelium. I think there can be little doubt that the lesions described by Dr. Davis are due to toxins reaching the terminals of the corneal nerves. It is possible that his type of punctate keratitis is an attenuated form due to a less potent toxin acting over a longer period of time.

DR. OSCAR WILKINSON, Washington, D. C.: I should like to ask Dr. Davis how he explains the ability to make a stain with fluorescein when there is no involvement of the corneal epithelium. It seems to me that if there is no involvement of the epithelium, one would not get a stain.

DR. W. THORNWALL DAVIS, Washington, D. C.: My presumption is that the fine spots take the fluorescein stain because the stain may to some extent be absorbed through the cornea, enough to effect a pathologic change of this type. That is the only explanation I can offer. Most careful study reveals no change in the epithelium.

OCULAR LIPID HISTIOCYTOSIS AND ALLIED STORAGE PHENOMENA. DR. PARKER HEATH, Detroit.

This paper will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. C. S. O'BRIEN, Iowa City: Our conceptions of disease are constantly changing. It is only a few years since we first heard of the reticulo-endothelium. The histiocytes, or fixed phagocytes, play an important rôle in tissue response. I am sure we have all noted in our study of pathologic processes under the microscope a great similar-

ity in the microscopic appearance of lesions, even though they are produced by widely different causes. One sees evidence of fatty changes in many diseases, but whether they are responsible for further changes, as Dr. Heath believes, or are simply evidence of degeneration, I do not know. It has been my opinion that they are end-processes and are due to degenerative changes.

Dr. Heath's classification, based on histopathology, may enable us to form certain groups, as he has done, classifying these isolated disease entities into one group, explainable on the same pathologic basis and accounted for by the disturbances in fat metabolism.

FIXATION AND VOLUNTARY NYSTAGMUS: A CLINICAL STUDY. DR. ALFRED F. LUHR and DR. JOHN L. ECKEL, Buffalo.

This article will be published in full in a later issue of the ARCHIVES.

ASTIGMATIC CHARTS VERSUS CROSSED CYLINDERS: RESULTS IN ONE HUNDRED OFFICE CASES. DR. WALTER B. LANCASTER, Boston.

The methods of using the astigmatic charts and the crossed cylinders are described in some detail. There is no final authority to which one can appeal. It can be demonstrated conclusively by the laws of optics and of visual physiology that each method gives valid results. This, of course, is borne out by clinical experience. The sensitiveness of both tests depends on the patient's powers of observation. If one method proves superior in a given case, it is because the patient is able to choose more accurately, promptly and consistently with this method than with the other one. The author's conclusion is that some patients do better with one method and some with the other. Hence, one should be able to make use of both. If limited to one, the author would take the astigmatic charts as more frequently giving more accurate, prompt and consistent results.

DISCUSSION

DR. EDWARD JACKSON, Denver: With the crossed cylinder, the objective certainty can be reached in a different way. Try to focus the meridians with the crossed cylinder, and when you think you have them focused, change the cylinder and try the patient again. In that way I test the meridians for astigmatism from three to six times at one sitting. The patient knows nothing about how you are turning the cylinder, does not know where the plus axis or the minus axis is and has no guide to decide where he should want the cylinder in the trial frame. He has no guide for sticking to his story; he simply has to depend on his sensation. When you get half a dozen tests of that kind which agree within 1 degree of each other, you can rely on the chosen axis for the cylinder with more certainty than on the ophthalmometer or the shadow test, which are purely objective. The same certainty is reached when the method is modified to get the strength of the cylinder required. I frequently find that patients, repeatedly tested, prefer something stronger than a cylinder of 1 diopter and something weaker than a cylinder of 1.25 diopters. It is a much more accurate way of deducing intervals of one-eighth than to use cylinders that

change by 0.125 diopter. By reasoning and repeating the tests, the crossed cylinder can be made to have the value and force of an objective test.

The first twenty years that I measured refractions I used a rotating chart. I then used the ophthalmometer, the shadow test and the crossed cylinder, with the eye under a cycloplegic, and post-cycloplegic testing. After trying all these methods, for the last ten years I have relied on the crossed cylinder for the correction of astigmatism and to determine the amount of astigmatism.

Dr. Sylvester J. Beach, Portland, Me.: The two methods of examination that have been discussed are not mutually exclusive. Neither can be neglected if extreme accuracy of refraction is desired. Disappointment in these devices may in part be due to a lack of understanding of their limitations. The astigmatic dial is unsatisfactory in irregular astigmatism. Some years ago I studied the appearance of the astigmatic chart as described by a series of patients, and found that a misleading degree of irregularity is unexpectedly prevalent. One type of asymmetry, for instance, will place the astigmatic axis in the upper hemisphere from 20 to 40 degrees from that below the horizontal. Such a possibility ought entirely to preclude the use of the half dial or astigmatic fan, which gives no clue to this discrepancy.

The weakness of the crossed cylinder lies in the fact that it has to be used in connection with some form of test card. Patients often prefer a glass that somewhat distorts certain letters by overemphasizing distinguishing lines. They are also confused by the difference between our test type and their ordinary printing, which is higher than wide and has the vertical lines accentuated. Moreover, as I have frequently pointed out, the acute-angled letters are a more sensitive test for astigmatism than round ones and those with right angles.

If trial characters for the crossed cylinders are intelligently selected and if too much reliance is not placed on the astigmatic chart in cases of irregular astigmatism, these two tests take their place among the most delicate and useful tests we have.

Detachment of the Retina: Guist Operation and Report of Cases. Dr. Hugh S. McKeown, Brooklyn.

This paper reviews the literature and cites the claims of various workers of from 10 to 50 per cent of cures by use of the thermocautery.

The Guist operation is the multiple trephining of the sclera over the area of the detachment, the treatment of the choroid by chemical cauterization (potassium hydroxide) and the liberation of the subretinal fluid.

The paper further reports the cases of all patients operated on during the past year, outlines the complications which occurred and advises how these may be prevented.

The author further reports that from 50 to 75 per cent of cures and improvements are being obtained.

Dr. Harry S. Gradle, Chicago: We have had less than 15 per cent good results over a long period of time, and from what I have seen of others I am sure that if we can maintain 15 per cent results

in detachment of the retina by surgical methods we should congratulate ourselves because that is nearly 15 per cent more than by any other method.

To my mind the etiologic import of the hole in the retina is questionable. There are undoubtedly numerous cases of detachment of the retina that have reattached spontaneously despite the presence of the hole. There are undoubtedly numerous cases of detachment of the retina in which there is no hole whatever. Of course when we say "hole" we have reference rather to the smaller tears in the retina than to the larger rents proceeding posteriorly from the ora serrata.

In certain cases of detachment of the retina the original Gonin method is entirely unsuitable. In case of limited detachment, with or without a localizable hole, the Gonin method means a certain percentage of chance for good results; but you may have detachment of the retina existing in a fairly limited area where the other form of operation is frequently successful. They are the type in which the Guist offers the best chance for success, but frankly, I am afraid of the Guist operation. I am afraid of a cautery that I cannot control; I am afraid of possible drainage of the eyeball; I am afraid of the large choroidal and subchoroidal hemorrhages that so frequently occur during this operation.

Trephining the sclera is not always easy; it depends entirely on the location in which you wish to trephine. It is so easy to perforate the choroid, and usually when that is done your operation is concluded for the day. So after exposing the area in which I wish to make the aperture through the sclera I incise the sclera with a flat scalpel, the same incision as in cyclodialysis; then I form a pocket 5 mm. in depth and 2 or 3 mm. each side, separating the choroid from the sclera. Into the pocket thus formed I insert the flat blade of the Herbert punch forceps and bite out a scleral button.

Dr. Daniel B. Kirby, New York: The sealing of any hole or tear which may be present to prevent the herniation and drag of the vitreous on the retina is important. This may be accomplished with a certain degree of good fortune by the Gonin procedure. It is accomplished with greater ease by the Guist method, which secures in addition cohesion of the reapposed retina over the entire area of detachment. It is not necessary so accurately to localize the tear or hole, as a number of trephine openings may be placed at or near the area of the sclera to which that portion of the retina will float when the subretinal fluid is released.

Dr. Peter Kronfeld, Chicago: At the last meeting of the German Ophthalmological Society in Leipzig, which I had the privilege of attending, Guist reported cures in as many as 77 per cent of his uncomplicated cases. Dr. McKeown gives 39 per cent, and Dr. Gradle 15 per cent. We have every reason to believe that Guist, McKeown and Gradle are men of equal skill. The difference must lie somewhere else. I have been doing the Guist operation for a year and a half (eight or nine cases) and I am still afraid of the dangers involved in this operation.

A serious complication is interference with a large blood vessel. We cannot say in which direction the vorticose veins go after they enter

the eye, and the vorticose sinuses vary a great deal in extent and location. I personally have seen quite a few serious hemorrhages during the operation.

Lindner has apparently given up the Guist method. When I saw Lindner early in July of this year he was greatly impressed by the good results that Weve and others reported with diathermic microcoagulation. The principle is the same—we are trying to surround the diseased portion of the retina by a row of artificial adhesions produced either by the potassium hydroxide stick, or according to the new method, by electrothermic coagulation, which in my opinion and that of others is a much simpler and safer method.

UNIOCULAR MYOPIA. DR. JONAS S. FRIEDENWALD, Baltimore.

A statistical study of the distribution of refractive errors shows an excess of cases of high myopia over those with high hyperopia. As most body measurements, such as height and weight, when charted statistically yield a curve symmetrical about the mean, it has generally been concluded that the excess of high myopia is due to some factor other than the normal variations in the size of the eyeball. This additional factor has generally been assumed to be the effect of reading or other close application of the eyes which results in eyestrain.

Persons who are myopic in only one eye or who have a much higher myopia in one eye than the other are favorable subjects on whom to test this hypothesis, since, in general, they read with their less myopic eye or at least have a defective fusion sense, and therefore do not exercise convergence in reading. A statistical study was therefore made to investigate the various degrees of myopia occurring among all the uniocular myopes found among thirteen thousand successive cases seen in office practice. The results show that these cases fall into three groups: (1) those in which the myopia may be attributed to the normal variation in the size of the eyeball, which may be called normal myopia; (2) those in which the myopia is the result of injury or severe inflammation of the eye in childhood, which may be called secondary myopia, and (3) a group of cases that has its highest frequency at about 20 diopters, and which is distributed symmetrically about this mean; this may be designated as pathologic myopia.

Secondary myopia and pathologic myopia are relatively much more common among uniocular myopes than among binocular myopes. There is no evidence from this study that any form of myopia is related to eyestrain.

DISCUSSION

DR. MEYER WIENER, St. Louis: In a large proportion of cases hereditary predisposition unquestionably exists. We must not confound the terms "hereditary" and "congenital" under the former. We must consider the future of the eye as the tendency to develop to that which it was potentially ordained at the time of conception. This is not necessarily a fixed thing, but can be altered by various factors. We do know that if we take two bowls of tadpoles of the same age and feed one with ordinary food, giving the other the same with the addition of pituitary extract, the second lot will attain a much larger growth, even though both groups come from the same hatching. Therefore, it is

fair to assume that the hereditary tendency to stretching of the sclera, or progressive myopia, may be altered or averted under proper conditions.

In a group of forty-one cases of uniocular myopia observed by me in the last few years, eleven showed an increase in the refraction of one or both eyes over a period of from one to twelve years, varying from 0.5 to as much as 4 diopters. In four of them the refraction increased so that the myopia was binocular at the last observation. One would naturally assume that a large proportion of the patients with binocular myopia in whom the degree was different in the two eyes had been either hyperopic in both eyes or hyperopic in one eye and myopic in the other, at an earlier period. I am inclined to believe that monocular myopia cannot be considered as a separate entity from the binocular variety.

DR. E. M. JOSEPHSON, New York: In checking over the work of Baillart on the retinal circulation, I observed with particular frequency in the cases of monocular myopia that it was possible by comparatively slight pressure on the myopic eye to cause the appearance of the arterial pulse and then obliteration of the vessel, before any change could be effected in the veins. Apparently the venous pressure was higher than the arterial pressure, a state of affairs that could hardly be rational. No doubt the phenomenon is a paradox, due to a difference in the structure of the vessels and a difference of response to stretching of the eyeball by the disease process. It is noteworthy that the amount of pressure required to secure the obliteration of the arterial lumen was very slight, hardly greater than the pressure exerted by the extrinsic muscles of the eye. This observation might serve to throw some light on the progressive nature of myopia. Incidentally, the tension in these cases was within the range of normal.

COMPLETE DISCISSION OF THE CRYSTALLINE LENS. DR. CONRAD BERENS and DR. OLGA SITCHEVSKA, New York.

Complete discission of the lens has been criticized by eminent surgeons, but our experience in the majority of cases apparently does not support their conclusions.

The technic to be described differs from Ziegler's original technic in that a subconjunctival incision at the limbus has been substituted for the corneal incision. A single straight incision through the lens is a simpler procedure than the inverted V-shaped incision and has apparently yielded comparable results.

Preliminary iridectomy, which we formerly combined with complete discission, has not been performed on patients operated on in recent years. Possibly the results have been less successful in certain cases because preliminary iridectomy has not been performed.

The results of complete discission with a single straight incision have been good with few exceptions. Postoperative reaction has been diminished in young patients, and the necessity for repeated operations has decreased, especially in the last five years, when the incision in the anterior and posterior capsules has been made longer and the single straight incision through the lens has entirely replaced Ziegler's inverted V-shaped incision.

Complete discission of the lens has been employed successfully in treating congenital, juvenile and traumatic cataracts, and is also useful for the removal of the lens in myopia of high degree.

The results of operations on human eyes and on animal eyes are tabulated.

We believe that complete discission of the lens offers the advantages of (1) fewer operations to obtain comparable results; (2) less reaction than anterior capsulotomy, (3) less likelihood of a secondary increase in tension and (4) a diminished danger of infection.

It would be unfortunate if, because of adverse criticism, this procedure were to be discarded without further trial.

DISCUSSION

Dr. E. C. Ellett, Memphis, Tenn.: Since the publication in 1921 of Ziegler's paper on through-and-through discission of the lens for soft cataract, I have made use of the method as the opportunity presented, with results that have seemed to me more satisfactory than those obtained by the method I had previously employed. The final visual result is probably not affected, but solution of the lens occurs with less disturbance and less subsequent interference than formerly. My experience is too small to enable me to say that it occurs any more rapidly.

Dr. Meyer Wiener, St. Louis: Sometimes after the discission, though rarely after the Ziegler through-and-through discission, we have found the tension increased; this we have been able to relieve with an epinephrine substitute or epinephrine packs. My experience with an epinephrine substitute has not been favorable in glaucoma, but this type of case is an exception.

Dr. V. M. Hicks, Raleigh, N. C.: The importance of complete mydriasis cannot be overemphasized, and in this case we have favored scopolamine over atropine because we have found that in a number of cases the continued use of atropine has given an allergic reaction. The lens is cut easily with a Ziegler knife in a sawing fashion, but I believe it is important that the cutting occur only on the downward stroke of the knife. In a few cases I have completed a crucial incision, but the horizontal cuts were made only through the anterior capsule. This seemed to hasten absorption, and there was no evidence of unusual reaction.

The success of the operation should not rest on the basis of vision. In considering the success of the procedure, only the amount of reaction produced and the facility with which the lens is absorbed, together with injury to the other intracellular structures, should be considered. Because of previous degeneration in the posterior segment of the globe, we do not always secure good vision. The type of operation selected cannot possibly alter this complicated pathology.

Dr. George Slocum, Ann Arbor, Mich.: For a number of years, indeed since Dr. Ziegler's operation first came to my knowledge, I have been practicing through-and-through discission for soft cataracts, and the results have been so uniformly satisfactory that I have never returned to the older technic. In the first case of cataract in which I operated by this method there was no reaction; the pupil was more

easily kept dilated; absorption was more rapid; there was no secondary glaucoma, and a second needling was unnecessary. In subsequent experience a second needling has been done frequently, but the other advantages first observed have been manifested in practically every case of soft cataract in which such an operation has been performed. Perhaps I have been fortunate, but in my cases there have been no complications. I have not done a preliminary iridectomy.

VISUAL FIELD STUDIES: PSEUDO UPPER FORM FIELD CONTRACTIONS.
DR. W. D. ROWLAND, Boston.

In a series of patients studied in the Diagnostic Clinic of the Evans Memorial Hospital, Boston, among 1,903 routine examinations of the eyes, 230 patients showed a contraction of the upper form or white field which was recovered by raising the upper lid. These recovered fields are named pseudocontractions.

Other factors affecting the tone or level of the upper lids are discussed, as well as the commonly observed functional and organic changes involving the lid and orbital structures. The more subtle conditions of facial expression, muscle tone, and the like, reflected from general bodily conditions and refractive errors, were frequently found to be etiologic factors.

The following conclusions are presented: 1. Contractions of the upper field may be due to a wide variety of conditions affecting the lids, orbits, intra-ocular structures, optic nerve or the visual pathways. 2. If normal field limits are obtained by a wider palpebral fissure, the pseudocontraction differentiates the phenomenon from altitudinal hemianopia. 3. A careful study of the patient may also discover metabolic or other faults having a contributing bearing on the field change. 4. Perimetry is suggested as a means of measuring and recording ptosis.

DISCUSSION

DR. JOHN N. EVANS, Brooklyn: In normal subjects the position of the lid bears a relation to the visual line, which relation changes not only with the position of the eye, but also with the position of the head. More complicated still must be the factors that influence these relations in patients who are suffering from various diseases and in whom the reclining, sitting or standing postures can conceivably produce wide variations in the position of the lid and the angle of the head. One might think that the head rest of the Ferree-Rand perimeter would be sufficient guarantee of an ideal position, but the facial angle of a small series of patients examined in this head rest was such that the head was tipped forward 15 degrees.

Dr. Edward Jackson measured the effect of elevation of the lid on two hundred patients (*Tr. Sect. Ophth., A. M. A.,* 1921, p. 73), and presented definite figures to show that in all but seven of the cases the eye moved forward when the lid was raised. This protrusion of the globe was 0.75 mm. in most cases, but varied from 0.25 to 1 mm. It was practically absent in people after 60 years of age.

From these few points it at once becomes apparent that the interpretation of the upper peripheral field can be made only after careful consideration of a great variety of factors.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Oct. 14, 1932

A. C. HUDSON, F.R.C.S., *President, in the Chair*

IRIDOTOMY FOR DISLOCATED LENSES. MR. A. C. HUDSON.

This patient showed congenital dislocation of the lenses, with vision less than 6/60 and dilated pupils. The first eye was operated on by iridotomy in 1920, and the second similarly in 1925. The patient now has 6/9 vision in each eye with plus 10 spheres. Iridotomy seems to be a safer operation than iridectomy downward, because of the danger of loss of vitreous in those cases. In the present case there was no indication for interference with the lens.

DISCUSSION

MR. F. A. WILLIAMSON-NOBLE: I agree with the remarks concerning iridotomy.

TWO CASES OF ARACHNODACTYLIA WITH DISLOCATED LENSES. MR. A. C. HUDSON.

These patients, brother and sister, and two other members of the family had congenital dislocation of the lens and very fine fingers. They all had deformity of the bones of the chest and an appearance suggestive of acromegaly, with very long heads and prominent jaws. To deal with the lenses efficiently I had a special needle made, which was bent at an angle of 135 degrees, to facilitate introduction from the nasal side, to be used in conjunction with a Bowman's needle from the temporal side. It is true that these congenitally dislocated lenses are resistant to absorption, but they do absorb in time.

DISCUSSION

MR. A. W. ORMOND: When this dislocation of the lens occurs there is probably some abnormality of growth. The boy exhibited showed no developmental abnormality until he was 6 or 7 years of age. The class of acromegaly has become wider, and these cases may well be included in it.

PEMPHIGUS OF THE CONJUNCTIVA. MR. A. C. HUDSON.

In this patient pemphigus came on immediately after a severe attack of measles, with a purpuric eruption of the chest and arms. There were sores in the mouth, which were possibly pemphigoid. Four months later the skin cleared, and the conjunctiva became involved. The patient is gradually becoming blind.

DISCUSSION

MR. M. S. MAYOU: I have a patient, a boy 6 years of age, who has a shrinking conjunctiva and keratinization. He is treated with

liquid paraffin three or four times a day, and the cornea has not become opaque. The difficulty with vision is due to keratinization of the cornea.

MYOTONIC PUPIL. MR. J. D. M. CARDELL.

In two patients, one a man and the other a woman, the pupils showed myotonic reactions in accommodation only. In the man the supinator jerks were sluggish, while the other tendon jerks were normal. In the woman all the jerks were increased, a very unusual occurrence.

DISCUSSION

MR. FOSTER MOORE: This kind of pupil was first described by me seven years ago, at a meeting of the Ophthalmological Society, under the term "nonsyphilitic Argyll Robertson pupil," because it did not respond to light, but reacted in convergence.

THE RELATIVE MERITS OF METHODS OF TREATING SENILE CATARACT. MR. A. C. HUDSON.

When I was house surgeon at Moorfields, twenty-five years ago, I was deeply impressed with the serious consequences that were apt to result from prolapse of the iris after simple extraction and from incarceration of the lens capsule and iris in the incision after combined cataract extraction. Shortly afterward I saw the operation of extraction with peripheral iridectomy performed by Hess, and this seemed to afford a safeguard against the accidents incidental to other methods. I forthwith adopted it, and have continued to use it as a routine procedure to the present day. The credit for the inception of this operation is due to H. Beckles Chandler of Boston. Mr. Hudson submitted an analysis of 485 consecutive cases, to show the complications and to give an opportunity for estimating the relative claims of extracapsular and intracapsular extraction. Incarceration of the lens capsule in the incision was not seen or suspected in any of the cases. It may be claimed that the same result can be obtained with complete iridectomy if the operation is deferred until the lens has been delivered and the iris replaced; but, even so, the great value of the preservation of the sphincter, from the points of view of appearance and function, cannot be gainsaid. I think that it has the further value of antagonizing the forward displacement of the iris toward the incision. Prolapse of the iris occurred in 30 cases or about 6 per cent of the total number. In 17 of the 30 cases of prolapse it was found possible to replace the iris, with or without peripheral iridectomy. The practicability of replacing the iris was, in many cases, dependent on the fact that the prolapse was of small size, being limited to the portion of the iris at one side of the iridectomy and going entirely beneath the conjunctival flap.

One must remember that the iris at the site of, and adjacent to, the prolapse will have become adherent, by its anterior surface, to the corneosclera peripheral to the cataract section, and that unless this adhesion is separated, reposition will be imperfect and will probably be followed by a recurrence of the prolapse.

Loss of tone of the iris from excessive stretching of the sphincter during delivery of the lens no doubt increases the liability to pro-

lapse, but probably the presence of a lens remnant behind the iris is the most important local factor. For that reason it is advisable to use homatropine hydrobromide before the operation, so as to facilitate evacuation of the lens, and to instil atropine at its termination to afford the lens débris as free a passage as possible forward through the pupil.

Loss of vitreous occurred in 3 of the cases (or 0.6 per cent), but this figure is of little value for the purpose of comparison with the results obtained in intracapsular extraction, because in most cases in which loss of vitreous either was anticipated or had occurred, a complete iridectomy was performed. The percentage loss of vitreous has therefore been calculated for all cases in which cataract extraction with limbal incision was performed during the period reviewed (from 1916 to 1932). This figure is 2.9 per cent for a total of 550 extractions.

As a safeguard against loss of vitreous in extracapsular extraction, special lid retractors are used.

In any case of cataract extraction in which living capsular epithelial cells are left behind, however good the union may be directly after the operation, sooner or later—probably within a year or two—vision will deteriorate, sometimes to a remarkable extent, not only from thickening of the capsular membrane but also from wrinkling of the membrane. For this reason in most of the cases a capsulotomy was performed, almost as a routine measure.

In cases in which a tough band of capsule had to be divided, a Ziegler's knife was used; in the case of a broad band, division of the whole band at once was not attempted, but a piecemeal division was carried out, adhesive small portions being picked up with the point of a knife and divided.

Glaucoma following capsulotomy is a serious condition, because after any incision into the eye, vitreous is liable to pass through the pupil into the incision, where its presence is likely to vitiate the success of the operation. For this purpose Herbert's sclerotomy is especially suitable.

DISCUSSION

Sir John Parsons: Sir John cordially thanked the president, on behalf of the Section, for the address, and said that he was able to support all that it contained.

Nov. 11, 1932

A. C. Hudson, F.R.C.S., *President, in the Chair*

A Case of Retinal Pigmentation. Mr. A. C. Hudson.

The patient has normal vision, 6/6 in each eye, the visual fields are not constricted, and there is no ring scotoma. The fundus contains an amazing quantity of pigment. Except for defective teeth, the man has had no disease since scarlet fever and measles in childhood. There were no visual defects and no consanguinity in the ancestry.

DISCUSSION

MR. J. H. FISHER: Obviously the retinal elements in this case are not impaired seriously, and the pigment is probably in the deepest layers of the retina, perhaps in the choroid too. There is no sheathing of the vessels or atrophy of the disk.

A CASE OF PEMPHIGUS. MR. J. ATTLEE.

One of the best treatments for pemphigus is said to be the intravenous injection of antimony and potassium tartrate. Since treatment with mild doses of radium the right eye has improved.

DISCUSSION

MR. J. H. FISHER: A patient had conjunctival pemphigus with shrinking of the conjunctiva and much vascularization of one cornea. Bullae developed on the skin and the mucous membranes of the nose and mouth, and I gave an unfavorable prognosis. A year later the patient contracted measles, and with this attack improvement in the pemphigus began. Since measles is not a serious disease, it seems almost justifiable to expose the patient with pemphigus to measles.

MR. M. S. MAYOU: Ten years ago I had a boy with pemphigus and typical essential shrinking of the conjunctiva whom I treated locally with paraffin. Corneal involvement did not occur. In many cases of so-called pemphigus, I believe that the diagnosis is incorrect.

MR. A. C. HUDSON: I have given the serum of patients convalescing from measles to patients with pemphigus, but it appeared to have no effects.

MULTIPLE CONGENITAL ABNORMALITIES OF THE EYELID, CORNEA, IRIS AND LENS. DR. A. J. BALLANTYNE, Glasgow, Scotland.

A girl, 6 days of age, has edema of the eyelids, a small notch at the junction of the middle and nasal thirds of the upper lid, a dermoid, 3 mm. in diameter, on the limbus at 4 o'clock, another dermoid between 6 and 7 o'clock, a subconjunctival lipoma in the lower and outer part of the eyeball, xerosis of the conjunctiva, a coloboma of the iris, up and out, and three bands of tendinous tissue stretched across the anterior chamber from one angle to the other. In the other eye there are an anterior polar cataract and a dimple of the nasolabial fold. The Wassermann reaction is negative in both parents. It is a first-born child, there is no consanguinity in the parents, and they have no illness. No instruments were used at delivery.

DISCUSSION

MR. M. S. MAYOU: The dermoids can be explained, I think, by imperfect closure of the eyelid during fetal life. I showed some time ago that in animals in which the conjunctival sac is divided into two parts, as in snakes, it always remains lined with a single layer of epithelium.

MISS IDA MANN: Recently I saw three eyes with bands stretching across the anterior chamber, these being largely adherent to the back of the cornea; at a deep level there was also a persistent pupillary membrane.

MILIARY TUBERCLE OF THE CHOROID. MR. G. PENMAN and MR. EUGENE WOLFF.

A girl, aged 8 months, had been ailing ever since an attack of diphtheria three months previously. She had a cough, and at the base of the left lung the percussion note was impaired, with bronchial breathing. The Mantoux test was positive. Below the disk the left fundus showed a yellowish, ill defined, rounded and slightly raised area, about two-thirds the diameter of the disk. This proved to be a choroidal tubercle. The child died, and at autopsy there was found to be a swelling of the choroid in the vicinity of the lesion. There was an infiltration with round and epithelioid cells and caseating areas which stained pink with eosin. Tubercle bacilli were present in large masses.

CASES ILLUSTRATING THE MODIFIED TOTI OPERATION FOR LACRIMAL OBSTRUCTION. MR. EDWARD D. DAVIS.

Though pleased with the results achieved with the modified Toti operation I find that the operation can be said to have failed in 25 per cent of my cases. In all the cases described in this paper the patients had already been treated by ophthalmologists before I saw them.

A girl, aged 20, had had obstruction of the right lacrimal sac for a year; the sac had been incised twice because of the presence of suppuration. When I saw her she had a fistula of the sac on the face. The nose was normal. After a Toti operation the wound healed by first intention, and the case is a success.

A man, aged 43, had had epiphora and dilatation of the sac for eighteen months. The middle turbinate was cystic. The Toti operation was successful.

A man, 52 years of age, had had suppuration of the left lacrimal sac of two years' duration. The sac had already been incised three times, and there was periostitis of the lacrimal groove, with atrophic rhinitis. The Wassermann reaction was negative. A fistula and suppuration of the sac were present. Toti's operation was a success.

In a woman, aged 23, lupus of the nose, involving both inferior turbinates, had been treated by excision and the cautery. Epiphora and lacrimal obstruction were present. West's operation was done, first on the right side and later on the left; at the second operation the procedure was repeated on the right. The operation on the right side was a success. On the left side a Toti operation was done for the suppuration, and for eighteen months the result was successful. Then the suppuration returned and persisted, notwithstanding the fact that pus was freely discharged through the artificial fistula within the nose. This suppuration has not yet ceased.

The two conditions most liable to be followed by inflammation of the lacrimal sac are lupus and atrophic rhinitis. English rhinologists named the modified Toti procedure "Mosher's operation," as it was more recently described and advocated by Prof. Harris Mosher of Boston.

(Mr. Davis described his procedure in minute detail.) The external operation is mechanically easier and more accurate than either the

West or the Polyak intranasal dacryocystotomy. The intranasal operation cannot be carried out satisfactorily in a narrow nose with a deeply concave atrium of the middle meatus or in the small nose of a child. I have now completely abandoned the intranasal method. Some cases in which the intranasal operation failed a modified Toti operation succeeded. In twenty-four of my cases in which the Toti operation was performed there were six failures; i. e., the sac continued to suppurate, and epiphora was present. The most successful cases were those showing a dilated sac, either with or without suppuration.

DISCUSSION

MR. G. H. HOWELLS: The operation that I have been carrying out for some time at Moorfields Eye Hospital is closely like that described by Mr. Davis, the chief difference being that I remove more bone than he does. I remove the anterior third of the turbinate intranasally, and in some cases it has been necessary to remove the anterior ethmoid cell over the area. After the operation the sac is irrigated on alternate days during the first fortnight, and later at increasing intervals. The results have been satisfactory in the nine cases in which I have operated, but it is too early to speak definitely about these operations, as none was done more than a year ago. The external operation seems to hold out hope of curing this condition.

MR. AFFLECK GREEVES: Is it necessary to remove the anterior part of the turbinate? Mr. Traquair of Edinburgh obtains good results without interfering with the turbinates.

MR. T. HARRISON BUTLER: I consider that a larger proportion of of these cases of obstruction of the lacrimal sac are due to ethmoiditis than Mr. Davis leads us to believe. I have met with the condition known as diverticulum of the sac.

MR. DAVIES, Cardiff, Wales: I have been doing the external operation for twenty years, and have never interfered with the nasal contents, though in some cases I have though that removal of the anterior turbinate might have been an advantage. I remove the whole lacrimal fossa. I have performed the operation about three hundred times, and it was successful in about 70 per cent of the cases. The chief reason for failure, nearly always, is cicatrization. I do not remember a case in which there was trouble in the nose.

MR. E. D. DAVIS: The size of the turbinate varies greatly in different persons. I remove it because I fear that adhesions may form between the middle turbinate and the opening of the sac. In my department at the hospital I see many cases of ethmoidal suppuration, but it is rarely complicated by disease of the lacrimal sac.

CORRECTION

The following discussion on "Light Streaks on the Retinal Blood Vessels," by Dr. W. H. Wilmer, Dr. H. F. Pierce and Dr. J. S. Friedenwald, which was read before the American Ophthalmological Society, June 29, 1932, was inadvertently omitted from the March issue.

DR. A. E. DAVIS: As first suggested by Loring, I am still of the opinion that the light streak on the retinal vessels is due chiefly to the refraction of light. Opponents of his theory contended that the carmine solution used in his experiment did not represent a true blood stream, which would diffuse the light too much to form a light streak. I repeated Loring's experiment, using the blood of a warm-blooded animal, and got results similar to those that he obtained with a carmine solution, which demonstrated conclusively, in my opinion, that the light streaks are caused chiefly by the refraction of light. The background largely dominates the whole picture, as the cases cited from Noyes attest. Any one who has viewed a retinal vessel traversing a choroidal coloboma can readily observe the brilliant light streak on the retinal vessels. On the other hand, when retinal vessels run in front of dark pigment spots in the choroid, only the faintest light streak appears on the retinal vessels.

Dr. Friedenwald stated, "I have never seen a light streak in a vessel over a coloboma. My understanding of a coloboma is that it contains no retina, therefore no retinal vessels." I may say that I have seen such vessels time and again, and de Schweinitz in the eighth edition of his textbook, page 343, stated, "In some cases the retina may be recognized as a translucent veil covering the defect, and the retinal vessels occasionally pass into the defect." Fuchs, in the eighth edition of his textbook, page 727, gives an excellent illustration of retinal vessels passing over a choroidal coloboma.

In cases of well advanced detachment of the retina of long standing, my observation has been that the light streak is very faint or altogether absent, even when the vessels lie at right angles to the source of light.

Book Reviews

The Measurement of Visual Acuity. By R. J. Lythgoe. With assistance by Dorothy E. Corkill and with a section by E. S. Pearson. Report of the Committee on the Physiology of Vision, Privy Council, Medical Research Council. Price, 1s. 6d., net. Pp. 85. London: His Majesty's Stationery Office, 1932.

It is difficult to define visual acuity, which changes from day to day and from minute to minute. In the discrimination of details, a part is played by physical, physiologic and psychologic factors. In the highest grade of visual discriminations—the form sense—the psychologic factors play a major rôle and obscure the physiologic ones.

Visual acuity varies with the illumination of the object and of the surrounds—the general illumination in which the subject sits—and with the adaptation of the eye.

It was once believed that two points separated by one minute of visual angle appear discrete. The images of these points lie about 0.0043 mm. apart on the retina, slightly more than the diameter of a foveal cone. It was thought that in order for the eye to resolve two dots, their images should have an unstimulated cone between them. This theory is no longer tenable. According to Hofmann (1920), the smallest visual angle is 34 seconds in the open air and 31 seconds in a lighted room.

The author has adapted the definition as the ability of the eye to discriminate separate points, using as a test object the letter C, with the breadth of the line and the gap one-fifth the diameter of the letter. This was made in nine sizes. Adopting an arbitrary definition as to what is meant by saying that a certain object is visible, he chose as the end-point 4.5 correct answers out of 8.

After a discussion of many current theories, particularly with reference to the effect of increasing illumination, a description of the apparatus used is given.

The subject sits in a cube, the interior walls of which are painted a dead matt white and are 2 meters square. The subject has an unobstructed binocular view of the test object through a small window. The interior of the cube can be lighted to any desired extent. A camera was adjusted to photograph the pupil by red light, and a source of light, in a separate housing outside the cube, was used to test the effect of glare.

The test object, placed with the gap in the letter pointing in one of eight directions, was exposed by a revolving shutter for one and six-tenth seconds in every nine and six-tenths seconds.

In the earliest experiments a long series of readings was taken in which the size of the test object was varied, and the number of mistakes measured at each size. When the results were plotted, a sigmoid curve was obtained of the type met with in the biologic laboratory, such as the curve relating age to size of an animal.

Many of the older workers claimed that visual acuity comes to a maximum at comparatively low illuminations. The author found it to

improve up to, and probably beyond, 1,275 equivalent foot-candles, providing the surrounds are adequately illuminated, an increase not due to contraction of the pupils, but to increased sensitivity of the retina.

He found a progressive increase in visual acuity until the surrounds are from one-one hundredth to one-tenth as bright as the object, after which there is a falling off which becomes accentuated when the surrounds are brighter than the test object. This improvement is partly due to contraction of the pupil.

The results of the study of the effect of glare and of photography of the pupil are treated adequately.

In this report, Dr. Lythgoe, with the assistance of Miss Corkill in the experimental work and of Mr. Pearson in the statistical work, has made an extremely valuable and quite definitive contribution to the study of a complex problem.

JOHN B. LYNCH.

Mirrors, Prisms and Lenses. By Prof. James P. C. Southall. Third edition. Price, $4.50. Pp. 771. New York: The Macmillan Company, 1933.

This book covers essentially the work in geometric optics as given by the author in his courses at Columbia University. To some extent in the previous edition, but especially so in the new edition, important contributions to physical and physiologic optics have been included. In fact, this additional material enlarges the book by 140 pages over the preceding edition.

In the 18 chapters, the author covers everything of interest to the ophthalmologist with reference to the action of mirrors, prisms, lenses and combinations of these. Reflection, refraction, dispersion and correlated phenomena by simple and compound optical systems, including the human eye, are adequately treated and generally with a thoroughness not encountered in similar works. After building up elementary working formulas for different optical agents, the author proceeds to unify all into a few general principles. For example, he shows how by the application of the principle of reduced "vergences" it is possible to obtain a single, simple formula for conjugate foci, applicable to any symmetrical optical instrument, including mirrors, refracting surfaces and lenses.

To aid the student the author uses both the "ray method" and the "curvature method" in his explanations. An abundance of diagrams, about 350, including some photographic plates, amply illustrate the text. Appended to each chapter, save the last two, are a goodly number of problems which illustrate and frequently amplify the material covered in the preceding chapter.

Of particular interest are the sections dealing with the fundamental principles of some ophthalmologic instruments, e. g., the keratometer, the ophthalmometer and the Snellen charts. The section dealing with color vision, stereoscopic vision and stereoscopic projection, binocular fixation, perception of depth and related topics presents these matters in a brief, stimulating manner. Especially noteworthy is a critical analysis of the visual acuity and the resolving power of the eye with reference to the phenomenon of diffraction.

The book is a classic in the best sense of the term. It is an authoritative and comprehensive book to which the ophthalmologist can always turn for reference and enlightenment on any phase of "optics" involved in his practice.

JOSEPH I. PASCAL.

Studies from the Third Division of the Anatomic Institute of the Imperial University of Kyoto. Edited by Prof. Seigo Funaoka, Director. Series C. Experimental Investigation of Tuberculosis. Part 3. On Phosphatase: The Therapeutic Use of Phosphoric Acid Esters in Tuberculosis and Trachoma. Pp. 111. Kyoto: Anatomic Institute of the Imperial University of Kyoto, 1933.

In this volume are several articles of interest to ophthalmologists. Professor Funaoka, having produced peritoneal tubercles in rabbits, convinced himself that the intravenous administration of calcium-fructose-monophosphoric acid exercised a favorable influence on the absorption of these lesions. Being impressed by the similarity of the histologic pictures of trachoma and tuberculosis of the lymph nodes, he proceeded to treat a series of twenty-three patients with trachoma with calcium-fructose-monophosphoric acid. The drug is administered either intravenously or subcutaneously, in amounts of from 0.13 to 0.25 Gm., and is given at intervals of two or three days. Several of Funaoka's colleagues also report on their results of similar treatment, so that, in all, reference is made to ninety-five cases. The conclusions of all these investigators are practically identical and may be summarized as follows:

The subjective symptoms were entirely relieved after ten injections, improvement being noted after three. Hyperemia and conjunctival thickening diminished or disappeared. Granulations and papillary hypertrophy disappeared entirely after from twelve to thirty injections. Pericorneal infiltration and the blood vessels making up pannus formation also diminished after from nine to twenty-three injections. In some cases corneal phlyctenules disappeared after from six to fifteen injections. Scars were little influenced by the drug.

The volume also contains the reports of two cases of recurring vitreous hemorrhage (periphlebitis retinae tuberculosa) in which favorable results were obtained after a series of injections of calcium-fructose-monophosphoric acid.

O. P. PERKINS.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217, Faubourg St. Honoré, Paris.
Secretary-General pro tem.: Dr. A. Churchill, 66, Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Frank H. Crawley, 5 Fitzwilliam Pl., Dublin.
Place: Dublin. Time: July, 1933.

Ophthalmological Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgoam Road, Bombay 4.
Secretary: Dr. C. R. Athawale, Bhatwadi Girgoam, Bombay 4.
Place: A. M. C. Free Ophthalmic Hospital Bldg., Poi Bawadi Parel, Bombay 12.
Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.
Place: London. Time: May 11-13, 1933.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Place: Oxford, England. Time: July 5-8, 1933.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., W. 1, London.
Place: London. Time: June 9, 1933.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: June 26, 1933.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: Aug. 3-5, 1933.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Frederick H. Verhoeff, 82 Commonwealth Ave., Boston.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Milwaukee. Time: June 12-16, 1933.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time: Sept. 18-22, 1933.

American Ophthalmological Society

President: Dr. Thomas B. Holloway, 1819 Chestnut St., Philadelphia.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Washington, D. C. Time: May 8-10, 1933.

SECTIONAL

New England Ophthalmological Society

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. R. Secoy, 1802 Hewitt Ave., Everett, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary: Dr. William A. Wagner, 200 Carondelet St., New Orleans.
Place: Richmond, Va. Time: November, 1933.

Southwestern Michigan Triological Society

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids.
Secretary-Treasurer: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. J. J. Cohane, 59 College St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: December 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Ray Connor, 28 W. Adams Ave., Detroit.
Secretary: Dr. Ralph B. Fast, 136 E. Michigan Ave., Kalamazoo.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Anaconda. Time: July 11, 1933 (meetings are held semi-annually).

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: O. S. Hodges, 388 Pearl St., Beaumont.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. T. Hasler, 290 W. Center St., Provo.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Swart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. G. E. Clay, 384 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. D. Andrews, 33 Allen St., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Justin M. Waugh, Cleveland Clinic, Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Valin R. Woodward, Flatiron Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave. ,Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 312 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Elks Temple. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Karl N. Victor, Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month. Annual meeting, Jan. 10, 1933.

Milwaukee Oto-Ophthalmic Society

President: Dr. Samuel G. Higgins, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. P. E. Bousquet, 66 Sherbrooke St., West, Montreal, Canada.
Secretary: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Eugene Orr, 706 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. Seventy-First St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. L. G. Howard, 532 First Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. W. E. Munroe, 277 Alexander St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Max W. Jacobs, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. George N. Hosford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey, Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. J. L. Walter, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 504 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 504 State Tower Bldg., Syracuse, N. Y. N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, Medical Arts Bldg., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.